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# GENE MECHANISMS AND BEHAVIOR

JOHN L. FULLER

Division of Behavior Studies, R. B. Jackson Memorial Laboratory,
Bar Harbor, Maine

The demonstration of intraspecific differences in behavior which are due to genetic factors has been demonstrated by several workers. A bibliography of such studies up to 1946 has been given by Hall (1947). In animals such as the rat and dog, variability has been clearly shown in the motivational and temperamental aspects of behavior, conditioned reflex formation, and in the type of social relationships established (Scott, 1950; Searle, 1949; Stockard, 1941; and Thorne, 1944). It is possible that there are also differences in perceptual ability, in rate of learning, and in the ability to analyze complex problems; but difficulties in excluding the effects of emotional upsets in test situations make it hard to obtain conclusive proof. These results of animal studies are in some disagreement with the results of studies on human identical twins. A recent text on human genetics (Stern, 1949) summarizes many researches with the statement that the balance of evidence favors a greater genetic effect on "intelligence" as measured by standard I. Q. tests than on "temperament" as determined by objective rating scales. The contradiction may be more apparent than real, and further studies may reconcile the discrepancies. It should not be necessary to belabor the point that we are dealing with a field in which rigorously controlled experimentation is difficult. Behavior traits are highly modifiable by the environment, and environmental effects may be very subtle and difficult to detect. There is, however, no need to be excessively pessimistic. Experiments such as those of Scott (1950) are extending our knowledge of the range of phenotypic variation which a particular genotype will permit. Sociobiological and psychobiological studies of this kind may well have important consequences in education and social thinking.

To complement behavior studies, however, there is place for research on the processes which lie between the transmission of genes from parent to offspring and the appearance of a characteristic behavior pattern. It is a far cry from minute particles of nucleo-protein in an egg or sperm to behavior described in terms of responses of aggression, timidity, courtship, or the ability to solve differential equations. To some it may seem pre-

sumptuous to attempt to bridge this gap at the present time. We are a long way from understanding how genes control relatively simple metabolic processes, while attempts to find the organic basis of psychological phenomena have fallen short of the rosy hopes of a host of investigators. Nevertheless, I believe that there is value in a brief survey of our present state of knowledge which may point the way to profitable avenues of investigation.

Studies of gene control of the chemical characteristics of microorganisms, plants, and animals (Beadle, 1946; Catchside, 1950; Lincoln and Porter, 1950; Wright, 1941), and on gene mechanisms and morphogenesis of higher forms (Dunn, 1949; Gluecksohn-Schoenheimer, 1945; Grüneberg, 1947) are also pertinent to the relationship of genes to behavior. Physiological psychology is active with ideas concerning the energetics of human behavior (Freeman, 1948); the description of brain function in terms of the unities found through factor analysis (Halstead, 1947); theories of feedback and servo-mechanisms which have led to a new scientific field called cybernetics (McCullough, 1950; Wiener, 1948), and attempts to interpret the laws of behavior in terms of neurophysiological principles (Hebb, 1949; Lashley, 1949). Paralleling such theoretical approaches are many experimental studies on brain-operated animals covering emotions (Bard and Mountcastle, 1948; Speakman and Babkin, 1950), delayed response (Pribram, 1950), learning (Lashley, 1944) and a great deal of interest in problems of nerve metabolism. The effects of hormones on behavior have been admirably summarized recently (Beach, 1948; Collias, 1950), so that this possible means of genetic control of behavior can be objectively evaluated. All of these developments have an influence on our way of thinking about gene mechanisms and behavior.

Although behavior is a characteristic of the organism as a whole, and cannot be considered as a property of any one system, it is certain that the nervous system is central to the integration of responses into coherent patterns. Hence, it is profitable to emphasize the relationship of genes to the nervous system.

Expressed formally, gene action may affect behavior through direct or indirect action on the central nervous system. Direct effects include: control of the number of neural elements; determination of the interconnections of the neural elements; and modifications of the chemical and physical characteristics of neurons, so that they are more or less readily modified in the course of their functioning. In addition, there are secondary effects. Genes may determine the ability of an organism to utilize available nutrients, and, therefore, the energy available to the control system. Another type of control involves the effect of genes on the formation of chemical agents such as hormones, which affect the excitability of specific pathways in the central nervous system. Genes may also control the characteristics of exteroceptors (as in red-green color blindness), and thus influence the amount of information which the nervous system receives from outside the organism. Finally, they may influence the effectors, so that behavior

patterns requiring specific movements are facilitated or made impossible, and feed-back to the central nervous system is thereby modified. The direct effects of genetic sensory and motor defects on behavior are often obvious, and appear to be similar to those of the same defects of non-genetic origin. The indirect effects are primarily related to the fact that defective animals are at a great disadvantage when they are forced to compete. When protected from competition such defective individuals may adjust well emotionally and show no behavior deficits which are not clearly related to their primary disability. In the remainder of this talk we shall be primarily concerned with evidence for effects upon the functions of the central nervous system.

A complete case must include a demonstration that a particular physical character is gene controlled, and that a particular behavior pattern is a unit which is uniquely determined by the physical character. No such case is now known except for pathological conditions.

Turning to the direct effects of genes on the nervous system, one would expect that an increase in the number of neural elements would increase the flexibility of the control system and allow it to respond more selectively to variations in input signals. In general the brain-weight/body-weight ratio of vertebrates is positively correlated with psychological complexity (Lashley, 1947). No marked relationship between brain mass and behavior has been found within a species, though the work of Spitzka and Basset as quoted by Lashley indicated, in men and rats respectively, a tendency for superior performance to accompany increased brain weight/body weight ratio.

There are many cases of neurological defects in genetically controlled strains of animals. Examples include ataxic and epileptic rabbits, ataxic pigeons, waltzing, shaker and other choreic mice. (For details see Riddle, 1917; Nachtsheim, 1939; Sawin, Anders and Johnson, 1942; Grüneberg, 1943). In some cases (e.g. rabbit ataxia, Anders, 1945) these behavior mutants suffer from deficiencies in specific tracts of the nervous system. In the waltzing mouse no specific defect has been yet demonstrated as an essential component, although most waltzers have pathological conditions in the region of the acoustic nerve. In still other cases (rabbit epilepsy) the disorder is classed as functional because no structural pathology is known. These mutants may also differ in their general level of activity. For example, the waltzing mouse is extremely active and the males are recorded as very aggressive. Waltzing and shaker mice have good vitality and fertility, but other defects, such as rabbit ataxia, are semi-lethal and result in progressive deterioration and death before reaching the age of reproduction. It is unwise to draw too strict a line between the disorders characterized by known pathology, and those which are called functional, since the latter undoubtedly involve derangement of structure at a finer level, colloidal or molecular.

There are few quantitative studies on the nervous system of different genetic strains of animals. Smith (1928) has described variations in the

number of mitral cells in the olfactory bulbs of two strains of the albino rat, but he makes no reference to any behavioral differences which might arise from this condition. In general, it may be stated that careful quantitative studies almost always show great individual variability in the nervous system. This is well shown by a quotation from Lashley and Clark (1946) regarding their work on the structure of the cerebral cortex of the spider monkey: "There is scarcely a quantitative histological character which does not vary by 25 to 100% in local regions." These observations were made on presumably normal specimens, but no genetic studies were undertaken; nor is the behavioral significance of such quantitative morphological differences clearly established. A certain amount of structural variability occurs in strains of mice inbred for upwards of 100 generations. Characters such as the distribution of pigment rings in the tail and ventral spotting retain marked variability in essentially homozygous strains (Murray and Green, 1933). Perhaps the differences in cell arrangement of the cerebral cortex are produced by random mechanical factors rather than by gene differences. The research necessary to show the origin of this variability should definitely be undertaken.

In summary, therefore, the existence of individual differences in the structure of the central nervous system is well established, but their importance for differences of temperament, social behavior and intelligence is unknown, and genetic control has been shown only for gross neurological defects.

Genetic control may also operate through the production of variations in the autonomic nervous system. Lacey (1950) has demonstrated that individuals have characteristic modes of physiological response to stressful situations. One person may react chiefly by a rise in systolic blood pressure, another by changes in heart rhythm, and still another by changes in skin resistance. The important role of the autonomic system in learning simple habits has been shown by Solomon and Wynne (1950) who found that sympathectomy in dogs retarded the formation of an avoiding response. It is likely that feed-back from autonomic effectors is one of the factors influencing the modifiability of the central nervous system. Various workers in the field of individual physiology have postulated that genetic factors produce the individuality (Wenger, 1948; Jost and Sontag, 1944). Recent studies on pure breeds of dogs have established the fact that each breed has a characteristic physiological profile, which appears to represent differences in autonomic functioning (Fuller, in press). All of this evidence suggests that genetically controlled autonomic patterning may affect learned behavior, but intensive individual studies, in which all variables are controlled, are needed to confirm the hypothesis.

The question of genetic effects on the characteristics of the individual neurons leads to a consideration of individual biochemistry and metabolism (Keys, 1949; Williams, 1946). Recently the hypothesis has been put forward that alcoholism may be produced by a hereditary inability to utilize specific nutrients, and the term "genetotrophic disease" has been intro-

duced to describe such cases (Williams, Berry and Beerstecher, 1949). For many years we have known of phenyl-pyruvic amentia, a type of hereditary feeble-mindedness attributable to the failure of a specific enzyme to be formed in the brain. Here, again, we deal with a gross disorder sharply separated from the normal. Utilizing another gross deficiency, Koch and Riddle (1919) found that the brains of ataxic pigeons showed marked chemical deviation from normal, and the brain of the ataxic rabbit has been shown to be deficient in free amino acids (Roberts, Frankel and Harman, 1950). In chemical studies, as in anatomical ones, it is relatively easy to find evidences of genetic factors operating to produce gross pathologies but the significance of variability in the normal range has not been shown.

A promising tool for investigating the physiological genetics of behavior is the sound-induced convulsion characteristic of certain strains of mice when they are stimulated by a loud, high-pitched sound. Susceptibility to these seizures is definitely a genetic trait (Hall, 1947; Witt and Hall, 1949; Fuller, Easler and Smith, 1950), and is not merely a function of some extrinsic factor such as middle ear infection (Miller and Zamis, 1949). Animals of high and low susceptibility strains appear essentially normal in ordinary laboratory situations, although the highly susceptible strain is considered slightly less vigorous. The DBA strain, a highly susceptible strain, is also high in mammary tumor incidence, but other high mammary tumor strains have been found to be resistant to seizure. Young DBA mice, when exposed to sounds above 5000 cycles frequency at intensities near 100 decibels, usually go into a fit of wild running which terminates in a convulsion, often fatal. Ginsburg, surveying the literature on rat seizures, noted that many of the substances whose deficiencies increased susceptibility functioned in the body as enzymes. In a series of researches Ginsburg (1949) and his co-workers (Ginsburg, Ross, Zamis and Perkins, 1951) have investigated the physiological and chemical correlates of susceptibility. As a result of these studies Ginsburg has postulated that a contributing factor to seizure susceptibility is a deficiency in carbohydrate metabolism. This may be genic in the case of the DBA mouse, or nutritional in the case of the rat which is deprived of thiamine or magnesium. Deficiencies in enzyme systems due to genes are well known in *Neurospora* and relative deficiencies in the enzyme of a mammalian brain may be the cause of increased susceptibility to sound-induced seizures. Direct proof of this hypothesis still requires demonstration of metabolic differences in the nervous systems (or at least in certain parts of the nervous system) between susceptible and non-susceptible strains.

Closely allied to the gene-enzyme problem is the more general problem of the role of genes in protein synthesis. A physico-chemical theory of learning which has recently been developed by Katz and Halstead (1950), attempts formally to relate gene action and the type of change in the nervous system which accompanies learning. Their model of learning is based upon protein molecule denaturation by electrical fields set up during transmission of nerve impulses. The extreme diversity of the protein molecule

and the well-known specificity of molecules formed by different genetic systems, for example the Rh blood groups and the blood groups of cattle (Irwin, 1947; Stormont, 1950), give this theory a definite plausibility.

Neurons formed by the action of one set of genes might be composed of proteins which were more readily denatured than proteins formed by another set of genes. Psychologically this would result in more rapid learning. Furthermore, these denatured molecules might be highly stable, and the learned behavior would persist; or unstable, and it would rapidly become extinct. A hypothesis of this type involves imagination which goes beyond proven facts, but Katz and Halstead have suggested experimental means of testing the theory, and we may hope that it will stimulate fruitful research.

Turning to indirect effects of genes on the nervous system, the role of the endocrine glands must be considered. Beach's (1948) monograph enumerates many researches concerned with endocrine effects. If we take as an example the action of the sex hormones, it is obvious that what is essentially the same chemical substance produces quite different courtship behavior in various species of amphibia, birds and mammals. Conversely, even within the same individual, typical male and female behavior may be produced by appropriate treatment with androgens or estrogens. The control of sex behavior by hormones is more complicated than a simple channelling into male patterns by male hormones, and into female patterns by female hormones, but numerous experiments have shown that genetic potentialities which would normally never be expressed may be elicited by hormone administration. In experimental reversal of sex behavior, genetic specificity is not lost. The androgen-treated bitch behaves like a male dog, and not like some other species. The importance of the somatic substrate is further shown in experiments by Grunt and Young (1950) who castrated male guinea pigs of high and low sexual activity. Both groups fell to a very low level of sexual activity. When the subjects were placed on hormonal therapy with identical dosage, the amount of sex behavior increased in both groups, but high-drive animals returned to a high level and low-drive animals recovered only to their previous low level. A somatic factor of target organ sensitivity appears to be the most likely explanation for this finding.

Space will not permit an exhaustive consideration of genetics, endocrines, and behavior. Certainly, quantitative differences in hormone administration (Beach and Holz-Tucker, 1949) will have quantitative effects on behavior. The sexual activity of male rats increases with higher levels of testosterone injections. However, variation in tissue responsiveness to hormones appears to be widespread. The schizophrenic (Hoagland, 1950) has adrenals which are markedly unresponsive to ACTH, and a genetic factor has long been suspected in this disease. Speirs (personal communication) working at the Jackson Laboratory has found great variability in the eosinopenic response of various strains of mice when injected with corticoids. The mouse strains are also known to differ in their storage of adrenal lipids (Vicari, 1943). Although androgens are known to be important in producing so-called spontaneous fighting in mice (Beeman, 1947),

C3H and C57 black mice, which differ in fighting ability (Ginsburg and Allee, 1942) have similar levels of 17-keto steroid excretion (Karnofsky, Nathanson and Aub, 1944). Genes may affect behavior through control of the amount of hormones secreted, but probably a more important type of control is through modification of neurones and other target organs making them more or less sensitive to hormones, and thus altering the threshold of excitation of certain key circuits in the nervous system.

Another hypothesis for the mechanism of gene action on behavior is that genes affect the rates of physiological reactions in the nervous system. This theory is supported by evidence that brain wave frequencies have a hereditary basis (Lennox, Gibbs and Gibbs, 1945). A similar concept is familiar to students of growth, since morphological variability has been attributed to genetic control of rates of growth in specific centers (Huxley, 1932; Sawin, 1945). Excellent material for the study of gene-controlled time constants in the nervous system is found in the various mouse strains susceptible to sound-induced convulsions. It has been found that the latency of these seizures, that is, the length of auditory stimulation necessary to produce a convulsion, is shorter in strains with high susceptibility (Fuller, Easler and Smith, 1950). This led to the hypothesis that auditory stimulation induces at least two processes; one, excitatory, culminating in a violent spasm or convulsion; the other a process of accommodation which raises the threshold to convulsions. We do not have any definite evidence as to the nature of these processes. They may be primarily chemical, involving the production and hydrolysis of a substance like acetyl-choline (Freedman, Bales, Willis and Himwich, 1949); or physical, involving changes in the electrical properties of neurons which produce changes in the excitability of motor centers; or physiological, involving reverberating circuits with long persistence. The final result of prolonged loud auditory stimulation depends upon the outcome of a race between two opposing processes. If the excitatory process overtakes the accommodation process a convulsion occurs; if accommodation stays ahead, there is no convulsion, although preconvulsive behavior may be observed (figure 1). The line between convulsions and non-convulsions is sharply drawn, so that the outcome of the race can be accurately determined.

Fuller, Easler and Smith applied Wright's (1934) concept of developmental thresholds to their data on audiogenic seizure and concluded that "what appears to be a character of alternative expression is in reality a quantitative character which varies over a wide range." The general theory of heritability of threshold characters has recently been discussed by Dempster and Lerner (1950). These authors use the term *p scale* for the determination of a character in terms of its probability of appearance; for example the risk of a convulsion when a mouse is exposed to a standard auditory stimulus. The term *x scale* is used for the continuously variable factor which presumably underlies the character which can be directly measured on the *p scale.* The evidence is very strong that one underlying variable determining susceptibility to audiogenic seizures in mice is a rate

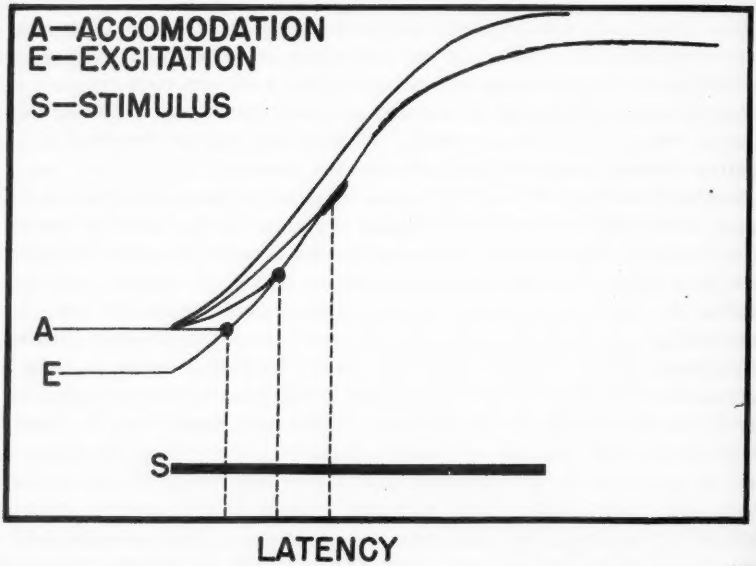


FIGURE 1. The relationship of stimulus, accommodation rate, and latency of convulsion in audiogenic seizures of mice. At the onset of stimulation, the level of excitation rises rapidly to a maximum. At the same time a process of accommodation starts at a rate depending upon the genotype of the mouse. The figure shows four different rates of accommodation; the slowest results in a very small latency; the fastest prevents the occurrence of a convulsion.

of accommodation which is inversely related to measurements of latency. Convulsion latency is remarkably constant under various experimental conditions in both rats and mice. Morgan (1941) and Wiener and Morgan (1945) found that latency was unaffected by intensity and quality of stimulation, or by cortical lesions, although these factors influenced the incidence of convulsions. Convulsions are always preceded by a period of wild running lasting from two or three seconds up to as much as thirty seconds. Following a suggestion of Dr. Ginsburg, strain A mice (34 days old at the beginning of the experiment) were exposed to auditory stimulation in the usual manner, but were forced to run by pushing them with a small paddle whenever they attempted to stay in one place. The animals were divided into two groups. Group I was scheduled: Agitation trial, 48 hours, non-agitation trial, 48 hours, agitation trial, until 8 trials were given. Group II was scheduled the same, except that the odd numbered trials were *"non-agitated,"* and the even trials were *"agitated."* No significant differences were found between the groups. Each animal served as its own control, and the data were analyzed for relationship of agitation to the seizure risk, to seizure latency and to the occurrence of the first convulsion. There was an indication that under the conditions of the experiment, susceptibility increased following the first convulsion. The results were:

| | Trials | Convulsions | First convulsion | Mean latency |
|---|---|---|---|---|
| Agitation trials | 92 | 55 | 19 | 62.1 ± 2.8 |
| Non-agitation trials | 92 | 38 | 7 | 63.3 ± 4.0 |

There is no difference in latency, although agitation significantly increases both convulsion risk of a given individual ($p < .01$) and the likelihood of a convulsion in a previously resistant animal ($p < .02$).

All this evidence indicates that the mechanism determining latency is near the end of the chain of events which culminates in a convulsion, and that it is relatively insensitive to experimental manipulation. To date, the genetic effect is the only variable known to modify convulsion latency in mice, though it is likely that experimental control could be produced by utilizing the same physiological means which are employed by the genes. Some function of latency seems to be the best candidate for the *x scale* variable.

Recent experiments (table 1) involving crosses between three strains of mice, DBA/2 with high susceptibility, A with moderate susceptibility, and C57BL with extremely low susceptibility confirm the threshold hypothesis which was originally based only upon crosses between the first and third strains. The adjusted mean latencies recorded in this table are based upon a probit transformation of the data, under the assumption that non-convulsers have a presumptive latency which cannot be directly measured, but can be calculated if a normal distribution of latencies is assumed. Both measures of latency are negatively correlated with convulsion risk, but bear no relationship to the incidence of fatal convulsions. A separate genetic and physiological system appears to be involved in protection against the stress of undergoing a convulsion. Although the hypothesis that genes control convulsive behavior and the outcome of convulsive behavior by affecting the relative rates of accommodation is strongly supported by these findings, this type of experiment does not isolate the specific processes which are involved. Direct physiological and biochemical observations will be necessary.

TABLE 1

AUDIOGENIC SEIZURES IN VARIOUS INBRED STRAINS AND THEIR HYBRIDS.

| Cross | Convulsion risk | Mean latency seconds | S. D. of latency | Adjusted latency | Deaths per convulsion |
|---|---|---|---|---|---|
| DBA × DBA | .92 | 35.8 | 12.5 | 38.3 | .87 |
| DBA × A | .91 | 35.6 | 16.6 | 39.4 | .21 |
| DBA × C57 | .76 | 52.1 | 9.7 | 56.5 | .59 |
| A × A | .11 | 61.8 | 12.2 | 75.7 | .00 |
| A × C57 | .045 | 54.6 | ...* | est. 74.6 | .57 |
| C57 × C57 | | | No convulsions | | |

*Too few cases to compute a standard deviation.

To summarize briefly, genes might act on behavior mechanisms at the level of organs, cellular arrangement or molecular structure. Gross organ defects influence behavior, but many behavioral differences are uncorrelated with easily observed structural differences. There is a great deal of evidence for individual differences in the cellular arrangement of supposedly normal nervous systems, but the genetic control of these variations and their significance for behavior have yet to be worked out. Variations in autonomic patterns have been shown to have a genetic basis and are important in behavior, but correlations within an individual organism have not been made.

At the molecular level there is a great deal of presumptive evidence that the sensitivity to specific hormones and other physiologically active substances varies between individuals and between genetic strains of mammals. Students of behavior should make wider use of these cases of strain differences turned up by biologists. In the case of sound-induced seizures of mice, there is evidence for some sort of neural deficiency in susceptible mice. This deficiency is not absolute but alters the time constants of excitatory and adaptive responses so that a stress situation produces an explosive breakdown in certain genetic types. These genetically determined time constants are much longer than those of single neurons, which are usually measured in milliseconds, or of the "brain waves" of the cerebral cortex, which are measured in cycles per second. Their very slowness makes them particularly convenient for chemical and physiological analysis.

It is still a long way from nucleoprotein molecules to the behavior patterns of mating or of problem solving. The gap can be closed only by detailed exploration of promising experimental materials—animals which are well controlled genetically and well understood developmentally, physiologically and psychologically.

These problems are difficult, but they have a peculiar fascination. Ten years from now it should be possible to give a much more definitive account of gene mechanisms and behavior—one which will be more instructive to the reader and more satisfying to the writer.

## LITERATURE CITED

Anders, M. V., 1945, The histopathology of a new type of hereditary loss of coordination in domestic rabbits. Amer. J. Anat. 76: 183-199.

Bard, P. and V. B. Mountcastle, 1948, Some forebrain mechanisms involved in expression of rage with special reference to suppression of angry behavior. Res. Pubs., Assoc. Res. Nervous Mental Disease, 27: 438-45. Williams and Wilkins, Baltimore.

Beach, F. A., 1948, Hormones and Behavior. 368 pp. Hoeber, New York.

Beach, F. A. and A. M. Holz-Tucker, 1949, Effects of different concentrations of androgens upon sexual behavior in castrated male rats. J. Comp. and Physiol. Psych. 42: 433-453.

Beadle, G. W., 1946, Genes and the chemistry of the organism. American Scientist, 34: 31-53 (also 76).

Beeman, E.A., 1947, The effect of male hormone on aggressive behavior in mice. Physiol. Zool. 20: 373-405.

Catchside, D. G., 1950, Physiological genetics. Ann. Rev. Physiol. 12: 47-70.

Collias, N. E. in Gordon, E. S., 1950, A symposium on steroid hormones. Univ. Wisconsin Press, Madison, xiv + 396 pp.

Dempster, E. R. and I. M. Lerner, 1950, Heritability of threshold characters. Genetics 35: 212-236.

Dunn, L. C., 1949, Some relations between mutations and abnormal development. R. B. Jackson Mem. Lab., 20th Comm. 79-91, Bar Harbor.

Freedman, A. M., P. D. Bales, A. Willis, and H. E. Himwich, 1949, Experimental production of electrical major convulsive patterns. Amer. J. Physiol. 156: 117-124.

Freeman, G. L., 1948, The energetics of human behavior. Cornell Press, Ithaca, viii + 344 pp.

Fuller, J. L. (in press), Genetic variability in some physiological constants of dogs.

Fuller, J. L., C. Easler, and M. E. Smith, 1950, Inheritance of audiogenic seizure susceptibility in the mouse. Genetics 35: 622-632.

Ginsburg, B. E., 1949, Genetics and social behavior, a theoretical synthesis. R. B. Jackson Mem. Lab. 20th Comm. 101-124, Bar Harbor.

Ginsburg, B. E. and W. C. Allee, 1942, Some effects of conditioning on social dominance and subordination in inbred strains of mice. Physiol. Zool. 15: 485-506.

Ginsburg, B. E., S. Ross, M. J. Zamis, and A. Perkins, 1951, Some effects of 1 ( + ) glutamic acid on sound induced seizures in mice. J. Comp. Physiol. Psych. 44: 134-141.

Gluecksohn-Schoenheimer, S., 1945, The embryonic development of mutants of the Sd strain in mice. Genetics 30: 29-38.

Grueneberg, H., 1943, The genetics of the mouse. Cambridge, Eng., 412 pp.
1947, Animal genetics and medicine. New York, Hoeber, 269 pp.

Grunt, J. A. and W. C. Young, 1950, Some effects of testosterone propionate on the sex drive of castrated male guinea pigs. Anat. Rec. 108: 103.

Hall, C. S., 1947, Genetic differences in audiogenic seizures. J. Hered. 38: 2-6.

Halstead, W. C., 1947, Brain and intelligence, Chicago, Univ. Chicago Press.

Hebb, D. O., 1949, The organization of behavior, a neurophysiological theory. Wiley, N. Y., xix, 335 pp.

Hoagland, H., 1950, Stress and the adrenal cortex with special reference to potassium metabolism. Psychosomatic Med. 12: 142-148.

Huxley, J. S., 1932, Problems of relative growth. London.

Irwin, M. R., 1947, Immunogenetics. Adv. in Genetics, 1: 133-160.

Jost, H. and L. W. Sontag, 1944, The genetic factor in autonomic nervous system function. Psychosomatic Med. 6: 308-310.

Karnofsky, D. A., I. T. Nathanson, and J. C. Aub, 1944, The urinary excretion of estrogens, 17-keto-steroids, creatine and creatinine in high and low mammary tumor strains of mice. Cancer Res. 4: 772-778.

Katz, J. J. and W. C. Halstead, 1950, Protein organization and mental function. Comp. Psych. Monog. 20: 1-38.

Keys, A., 1949, The physiology of the individual as an approach to a more quantitative biology of man. Fed. Proc. 8: 523-529.

Koch, M. and O. Riddle, 1919, Further studies on the chemical composition of the brain of normal and ataxic pigeons. J. Comp. Neurol. 31: 83-110.

Lacey, J. I., 1950, Individual differences in somatic response patterns. J. Comp. Physiol. Psych. 43: 338-350.

Lashley, K. S., 1944, Studies of cerebral function in learning. XIII, Apparent absence of transcortical association in maze learning. J. Comp. Neurol. 80: 257-281.

1947, Structural variation in the nervous system in relation to behavior. Psych. Rev. 54: 325-334.

1949, Persistent problems in the evolution of mind. Quart. Rev. Biol. 24: 28-42.

Lashley, K. S. and G. Clark, 1946, The cytoarchitecture of the cerebral cortex of Ateles: a critical examination of architectonic studies. J. Comp. Neurol. 85: 223-306.

Lennox, W. G., E. L. Gibbs, and F. A. Gibbs, 1945, The brain-wave pattern, an hereditary trait. J. Hered. 36: 233-243.

Lincoln, R. E. and J. W. Porter, 1950, Inheritance of beta-carotene in tomatoes. Genetics 35: 206-211.

McCullough, W. S., 1950, Machines that think and want. Comp. Psych. Monog. 20: 39-50.

Miller, D. S. and M. J. Zamis, 1949, Independent occurrence of audiogenic seizures and middle ear infections in inbred mice. Anat. Rec. 105: 76 (abst.)

Morgan, C. T., 1941, The latency of audiogenic seizures. J. Comp. Psych. 32: 267-284.

Murray, J. M. and C. V. Green, 1933, Inheritance of ventral spotting in mice. Genetics 18: 481-486.

Nachtsheim, H., 1939, Erbleiden des Nervensystems bei Saugetieren. Handbuch der Erbbiologie des Menschen. Vol. 5 Springer, Berlin.

Pribram, K. H., 1950, Some physiological and pharmacological factors affecting delayed response performance of baboons following frontal lobotomy. J. Neurophysiol. 13: 373-382.

Riddle, O., 1917, A case of hereditary ataxia in pigeons. Proc. Soc. Exp. Biol. Med. 15: 56-58.

Roberts, E., S. Frankel, and P. J. Harman, 1950, Amino acids of nervous tissue. Proc. Soc. Exp. Biol. Med. 74: 383-387.

Sawin, P. B., V. M. Anders, and R. B. Johnson, 1942, Ataxia, a hereditary nervous disorder of the rabbit. Proc. Nat. Acad. Sci. 28: 123-127.

Sawin, P. B., 1945, Morphogenetic studies of the rabbit. I. Regional specificity of hereditary factors affecting homoetic variations in the axial skeleton. J. Exp. Zool. 100: 301-329.

Scott, J. P., 1950, Genetic differences in social behavior of dogs. Am. Psychol. 5: 261-262.

Searle, L. V., 1949, The organization of hereditary maze-brightness and maze-dullness. Genetic Psych. Monog. 39: 279-325.

Smith, L. A., 1928, A comparison of the number of nerve cells in the olfactory bulbs of domesticated albino and wild Norway rats. J. Comp. Neurol. 45: 483-501.

Solomon, R. L. and L. C. Wynne, 1950, Avoidance conditioning in normal dogs and in dogs deprived of autonomic functioning. Amer. Psychol. 5: 264.

Speakman, T. J. and B. P. Babkin, 1950, Changes in behavior following frontal lobectomy in dogs and cats. Arch. Neurol. Psychiat. 63: 433-443.

Stern, C., 1949, Principles of human genetics. xi: 617 pp., San Francisco, Freeman.

Stockard, C. R., 1941, The genetic and endocrinic basis for differences in form and behavior. Wistar, Philadelphia, xx, 775 pp.

Stormont, C., 1950, Additional gene controlled antigenic factors in the bovine erythrocyte. Genetics 35: 76-94.

Thorne, F. C., 1944, The inheritance of shyness in dogs. J. Genetic Psych. 65: 275-279.

Vicari, E. M., 1943, The adrenal lipids of mice with high and low mammary gland tumor incidences. Anat. Rec. 86: 523-543.

Wenger, M. A., 1948, Studies of autonomic balance in Army Air Force personnel. Comp. Psych. Monog. 19(4): 1-111.

Wiener, H. M. and C. T. Morgan, 1945, Effects of cortical lesions upon audiogenic seizures. J. Comp. Psych. 38: 199-208.

Wiener, N., 1948, Cybernetics. Wiley, New York.

Williams, R. J., 1946, The human frontier. Harcourt, Brace, New York.

Williams, R. J., L. J. Berry, and E. Beerstecher, 1949, Biochemical individuality. III. Genotrophic factors in the etiology of alcoholism. Arch. Biochem. 23: 275-290.

Witt, G. and C. S. Hall, 1949, The genetics of audiogenic seizures in the house mouse. J. Comp. Physiol. Psych. 42: 58–63.

Wright, S., 1934, The results of crosses between inbred strains of guinea pigs differing in number of digits. Genetics 19: 537–551.

1941, Physiology of the gene. Physiol. Rev. 21: 487–527.

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# INTERSPECIFIC COMPETITION BETWEEN DROSOPHILA FUNEBRIS AND DROSOPHILA MELANOGASTER

DAVID J. MERRELL

University of Minnesota

In the "Origin of Species," Darwin developed the concept of natural selection as the mechanism of speciation. As one phase of natural selection, the competition of organisms with one another was cited. This competition is primarily the result of the high reproductive potential of all species compared to the environmental capacity to support them. The biotic environment determines the extent of the competition, which is therefore governed by the numbers of organisms present. Since the requirements of members of the same species are most similar, Darwin suggested that intraspecific competition would be most severe, with the competition lessening, the greater the differences between various groups in their demands upon the environment.

The studies of competition since this first rather general statement of the problem have led to certain theoretical and experimental conclusions. On the basis of theoretical equations, Lotka (1925) and Volterra (1926), (see also Ross, 1908), independently predicted that two types with identical needs and habits cannot survive in the same place if they compete for limited resources. If both types are found together in nature, they must differ in their ecology, or else there are fluctuations in the environment, favoring first one and then the other competitor. Intraspecific competition between mutants of the same species has repeatedly shown that the wild type genes will survive the mutant alleles in populations of *D. melanogaster* (Lutz, 1911; L'Heritier and Teissier, 1937; Reed and Reed, 1950). An unforeseen exception to this rule is to be found in the survival of two types because of the superiority of the heterozygotes over the corresponding homozygotes for inversions in *D. pseudoobscura* (Dobzhansky, 1947). The survival of more than one type maintains genetic variability in the population.

Experiments with interspecific competition between similar species have also demonstrated the validity of the theory (Gause, 1934; Crombie, 1947; Zimmering, 1948). Careful analysis of the instances in which both species survive has shown that they occupy slightly different niches (Gause, 1934; Crombie, 1947).

The importance of these results to evolutionary theory is considerable. Organic evolution has been marked by the repeated divergence of new lines ("adaptive radiation"), which have invaded new habitats, and also by the evolution of more "efficient" types, which have replaced existing forms.

The extent to which competition has been responsible for these trends can not be readily estimated, but the conclusion that it has been a factor seems inescapable.

## METHODS AND RESULTS

Since the pioneer work of Pearl (1926), Drosophila populations have frequently been used to study the dynamics of populations (L'Heritier and Teissier, 1937; Bodenheimer, 1938; Dobzhansky, 1947; Sang, 1950; and many others). In many early experiments, the flies were placed in a closed system, and the course of events was followed to the exhaustion of the food supply and the death of the flies. More recently, maintenance of the populations has been made possible by the use of population cages (Dobzhansky, 1947) or population bottles (Reed and Reed, 1948) to which food can be added at intervals.

L'Heritier and Teissier (1935) observed that mixed populations of *D. melanogaster* and *D. funebris* in population cages reached an equilibrium with a large population of *D. melanogaster* and a small population of *D. funebris*, no matter what their initial proportions. They noted the inconsistency of their results with the mathematical theories of competition and with the results of Gause. The observation that stocks of *D. funebris* survived longer in a bottle than a pure culture of *D. melanogaster* raised the question of whether this predominance of *D. melanogaster* in mixed populations would hold under all conditions.

Therefore, five mixed populations were started in population bottles with five males and five females of each species. The Oregon R strain of *D. melanogaster* and a local strain of *D. funebris* were used. The number of populations under study can be readily doubled at any time by attaching a bottle of fresh food to each bottle of the population unit. This procedure was occasionally followed to make additional populations available for study. The food consisted of the standard cornmeal medium (Demerec and Kaufmann, 1945) seeded with brewer's yeast. The temperature throughout was 20 ± 0.5°C.

The course of events can be followed in table 1 and figure 1. The data consist of samples of the mixed populations taken to determine the relative proportions of each species, and are not the total number of adult flies in the populations. Both species have coexisted in the population bottles for almost two years. The "equilibrium," however, is clearly not stable, but may undergo wide variations. These fluctuations can be correlated quite clearly with the age of the food. When fresh food was added, the proportion of *D. melanogaster* increased; as the food became older, the proportion of *D. melanogaster* declined. No food was added to two of the units, and the final populations were composed only of *D. funebris* before they died out. This result was confirmed with eight more mixed populations, a total of ten altogether. Addition of food before the extinction of *D. melanogaster* resulted in an increase of this species. If the food was added at regular intervals, and the counts made at about the same relative time in the cycle of food addition, a fairly stable equilibrium appeared to exist with *D.*

TABLE 1

COMPETITION IN POPULATION BOTTLES BETWEEN *D. MELANOGASTER* AND *D. FUNEBRIS*

| Day | Populations | Sample n | Percent *D. funebris* |
|---|---|---|---|
| 0 | 5 | ... | 50.0 |
| 14 | 5 | 771 | 5.3 |
| 21 | 5 | 1832 | 2.0 |
| 28 | 5 | 1470 | 1.9 |
| 37 | 5 | 566 | 33.0 |
| 54 | 4 | 195 | 16.4 |
| 61 | 4 | 123 | 93.5 |
| 72 | 2 | 138 | 94.2 |
| 84 | 2 | 262 | 27.5 |
| 109 | 2 | 40 | 67.5 |
| 174 | 4 | 491 | 4.0 |
| 207 | 4 | 341 | 6.5 |
| 247 | 4 | 444 | 6.3 |
| 271 | 6 | 455 | 3.5 |
| 315 | 6 | 670 | 7.2 |
| 347 | 6 | 763 | 3.1 |
| 387 | 6 | 472 | 11.0 |
| 452 | 6 | 116 | 12.1 |
| 567 | 3 | 202 | 8.9 |
| 649 | 4 | 100 | 30.0 |

*melanogaster* predominating. This situation has persisted for more than a year. However, during this period, two units became pure *D. funebris*, and another pure *D. melanogaster*, indicating the precarious nature of the equilibrium. The losses probably occurred more or less by chance when the population of the lost species had been reduced by unfavorable conditions.

The data show that an equilibrium does, in fact, exist between two species of the same genus living in a system with a volume slightly greater

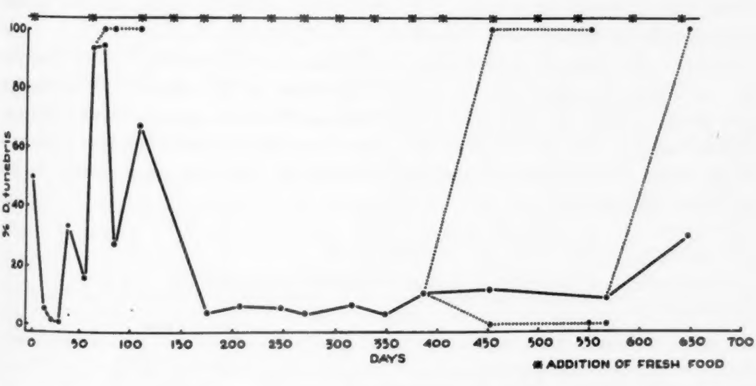


FIGURE 1. Competition between *D. melanogaster* and *D. funebris* in population bottles. No food was added to the two units reaching 100 per cent *D. funebris* at 72 days.

than a pint and drawing on the same basic source of energy. The next question to be considered is whether this result is an exception to the mathematical conclusions of Volterra and Lotka that species in direct competition with one another can not coexist.

It seems clear that even within this small system, the species are not occupying identical niches. In the first place, the system is neither closed nor constant, but fluctuates with the age of the food. The greater success of *D. melanogaster* on fresh food and of *D. funebris* on older food indicates a difference in their methods of utilizing the energy source. This difference is probably related to the differences between the micro-organisms on the fresh medium and those on the old medium, which have not yet been analyzed. The terms "fresh food" and "old food" are a simple means of referring to this undoubtedly more complex situation. Furthermore, if the food is not replenished, *D. funebris* does win out over *D. melanogaster.* Theoretically, on a constant supply of fresh food, *D. melanogaster* should eliminate *D. funebris,* but this experiment is not feasible. The food would have to be renewed so frequently that no *D. funebris* would emerge even without competition.

Since, in mixed populations, there was a delayed appearance of *D. funebris* pupae compared to their time of appearance in pure cultures, mixtures of the larvae of the two species were studied. To facilitate counting, petri dishes with a layer of yeasted medium were used. Five fertilized females of each species per petri dish were permitted to lay eggs together for a day. Controls were run for each species separately. After the adults were removed, the eggs were counted. Fortunately, the eggs of *D. melanogaster* have only two filaments while those of *D. funebris* have four. It is therefore possible to make a positive identification of the eggs laid by each species. Larval counts were not satisfactory because of the activity, the burrowing, and the lack of a simple means of classification of the larvae. However, the pupae are quite different in color and appearance so that the number of individuals of each species successfully completing the larval period could be readily determined.

Table 2 shows that, under these conditions, the presence of alien females had no apparent effect on the rate of egg deposition. The standard errors give an indication of the variability in the numbers of eggs per five females, but the sample size was also small. The *D. melanogaster* females appeared to lay eggs at a rather low rate, but it was the same in the controls as in the mixed populations.

TABLE 2

EFFECT OF ALIEN SPECIES ON EGG LAYING

| | Mean eggs/5 ♀♀ |
|---|---|
| *D. funebris* control | 87.7 ± 17.4 |
| *D. funebris* (with *D. melanogaster*) | 100.0 ± 17.9 |
| *D. melanogaster* control | 34.3 ± 12.4 |
| *D. melanogaster* (with *D. funebris*) | 33.0 ± 1.6 |

Table 3 shows the results of these experiments. Only one of the controls for each species was followed through this stage. The proportion of *D. melanogaster* eggs which reached the pupal stage did not differ significantly in the pure and mixed cultures and was over 90 percent in both cases. The presence of *D. funebris* larvae had no effect on the development of the *D. melanogaster* larvae even though they outnumbered the *D. melanogaster* larvae by at least three to one.

TABLE 3

THE EFFECT OF LARVAL COMPETITION

| | Control | | Mixed population | | | | Control | |
|---|---|---|---|---|---|---|---|---|
| | melanogaster | | melanogaster | | funebris | | funebris | |
| | n | percent | n | percent | n | percent | n | percent |
| Eggs | 24 | 100.0 | 130 | 100.0 | 445 | 100.0 | 131 | 100.0 |
| Pupae | 22 | 91.6 | 127 | 97.7 | 277 | 62.3* | 124 | 94.7 |

*Difference from the control is statistically highly significant.

On the other hand, the proportion of *D. funebris* eggs reaching pupation was definitely decreased in mixed larval populations. Almost 95 percent of the eggs formed pupae in the controls, but just over 60 percent pupated when *D. melanogaster* larvae were also present. Thus, under these conditions, *D. melanogaster* had an adverse effect on the development of *D. funebris.*

Figure 2 shows something of the nature of this effect. On the seventh day after egg laying began, pupation of *D. melanogaster* larvae was complete in both pure and mixed cultures. However, the first *D. funebris* pupae appeared in the control on the eleventh day, but not until the fourteenth day in the mixed populations. Even though no *D. melanogaster* larvae were present after the seventh day, not only was the rate of development of the *D. funebris* larvae retarded, but a third of them never pupated. It can be assumed that the losses of *D. funebris* in this case occurred primarily in the larval period. If there were no larval competition, but instead the *D. melanogaster* eggs hatched first and the larvae damaged or buried a certain proportion of the *D. funebris* eggs, then the time of pupation for *D. funebris* should have been the same in both control and mixed populations. Actually, the development of *D. funebris* averaged about three days longer in the mixed populations. In addition the hatchability of *D. funebris* eggs in the presence of *D. melanogaster* eggs laid at the same time was tested directly. Although the *D. melanogaster* eggs hatched sooner than those of *D. funebris* on the average, the young larvae did not affect the hatchability of *D. funebris* eggs significantly. The range in pupation time, even in the *D. funebris* control, indicates that the medium was not the optimum environment for the development of the larvae although 94.7 percent of the eggs in the control ultimately pupated. The above results

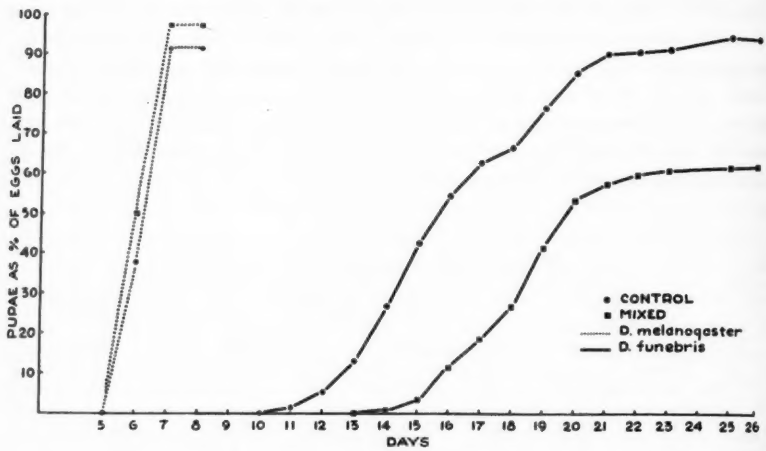


FIGURE 2. Competition between larvae of *D. melanogaster* and *D. funebris.*

with mixed larval populations show clearly that the two species do compete with one another directly to some extent.

Since the species are in direct competition, an investigation of the way in which both manage to survive was undertaken. The necessary clue lay in the fluctuations in the relative proportions of the species with the change in age of the food.

Two sets of experiments were run to test this difference. In the first, the half-pint bottles from a population unit were separated, and the larvae present were allowed to develop (table 4). The larvae were strained from the food in the old bottle and placed on fresh food. All the larvae from the old bottle turned out to be *D. funebris*. Adults of both species had had access to the new bottle for three days. These adults were removed and development allowed to proceed in the new bottle. Only *D. melanogaster* imagoes emerged. Thus, there was an apparent complete segregation, in this case, of the larvae. However, since at some time, a "new" bottle becomes "old," there must be a period when both kinds of larvae are present in the same food.

TABLE 4

LOCATION OF LARVAE IN A POPULATION UNIT

| | Funebris | | Melanogaster | |
|---|---|---|---|---|
| | ♂ | ♀ | ♂ | ♀ |
| Initial adult population | 3 | 16 | 11 | 12 |
| Adults emerging | | | | |
| Old bottle | 11 | 17 | 0 | 0 |
| New bottle | 0 | 0 | 95 | 107 |

In order to find out more about the differences between the larvae, a "Dobzhansky" population cage was started. The initial population was composed of *D. funebris.* This population was well established after two months, and 20 *D. melanogaster* were then introduced, 10 of each sex. Samples were taken from food cups of various ages to determine the proportions of each species present. The contents of a cup were transferred to two half pint bottles containing additional food so that favorable conditions for development of the larvae existed. The results are shown in table 5. Without regard to the adult population, which, of course, is a factor in the larval population, it can be seen that *D. funebris* inhabited both old and new food cups, but practically no *D. melanogaster* were obtained from the old food. In the cases in which both species were present

TABLE 5

LARVAL POPULATION IN CAGE

| Days after melano. added | Age of food | Funebris | | Melanogaster | | Age of food | Funebris | | Melanogaster | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | n | percent | n | percent | | n | percent | n | percent |
| 6 | 39 | 164 | 100.0 | 0 | 0.0 | 26 | 153 | 100.0 | 0 | 0.0 |
| 22 | 42 | 208 | 99.5 | 1 | 0.5 | 12 | 167 | 97.1 | 5 | 2.9 |
| | 42 | 141 | 100.0 | 0 | 0.0 | | | | | |
| 36 | 30 | 441 | 99.8 | 1 | 0.2 | 10 | 582 | 70.2 | 247 | 29.8 |
| 40 | 30 | 930 | 100.0 | 0 | 0.0 | 14 | 884 | 86.6 | 137 | 13.4 |
| 76 | 50 | 94 | 100.0 | 0 | 0.0 | 17 | 538 | 82.3 | 116 | 17.7 |

in a cup, almost all of the *D. melanogaster* had emerged before any *D. funebris* adults appeared. *D. melanogaster* was evidently able to maintain itself in the cage because of its faster rate of development on the fresh medium. However, this initial advantage did not persist, and eventually the food was suitable only for the development of *D. funebris.* The possibility that the females differ in their choice of sites for oviposition would, of course, also influence the results. However, the interpretation given above is probably essentially correct since the course of events was similar in the mixed populations in which the females had no choice because no food was added.

Lin (unpublished) has performed certain experiments which throw further light on the larval competition. He studied competition between just two larvae for a quantity of yeast insufficient for the normal development of both. If both larvae were of the same species, either *D. funebris* or *D. melanogaster,* they both failed to grow normally. However, if a larva of each species was present, the *D. melanogaster* larva developed normally, but the *D. funebris* larva did not. These results confirm the superiority of *D. melanogaster* over *D. funebris* in its ability to grow on yeast.

Table 6 and figure 3 contain the data on the adult population in the cage. The build-up of the *D. melanogaster* population in the cage was quite regular until by the 73rd day practically all the adults were *D. melanogaster.*

However, by that time, all except three cups were 39 days old or more. Table 5 shows that cups of this age could not support *D. melanogaster* larvae so that the sudden change in 11 days from an adult population consisting of almost 100 per cent *D. melanogaster* to one of almost 100 per cent *D. funebris* is not surprising. The addition of food cups at regular intervals should stabilize the proportions of the two species in the population.

TABLE 6

SAMPLE OF ADULT POPULATION IN POPULATION CAGE

| Day | Funebris | | Melanogaster | |
|---|---|---|---|---|
| | n | percent | n | percent |
| 0* | ... | 100.0 | ... | 0.0† |
| 22 | ... | 100.0 | ... | 0.0† |
| 42 | 152 | 98.1 | 3 | 1.9 |
| 47 | 125 | 86.8 | 19 | 13.2 |
| 51 | 150 | 75.0 | 50 | 25.0 |
| 55 | 158 | 69.7 | 72 | 31.3 |
| 61 | 57 | 37.5 | 95 | 62.5 |
| 68 | 28 | 31.8 | 60 | 68.2 |
| 73 | 0† | 0.0 | 58 | 100.0 |
| 77 | 2 | 18.2 | 9 | 81.8 |
| 79 | 6 | 54.5 | 5 | 45.5 |
| 83 | 20 | 100.0 | 0† | 0.0 |
| 87 | 13 | 100.0 | 0† | 0.0 |
| 90 | 49 | 81.7 | 11 | 18.3 |
| 97 | 41 | 97.6 | 1 | 2.4 |
| 99 | 36 | 90.0 | 4 | 10.0 |
| 101 | 30 | 66.7 | 15 | 33.3 |
| 104 | 45 | 49.4 | 46 | 51.6 |
| 107 | 37 | 35.2 | 68 | 64.8 |
| 110 | 12 | 7.8 | 142 | 92.2 |
| 115 | 35 | 24.3 | 109 | 75.7 |
| 121 | 15 | 8.3 | 166 | 91.7 |
| 125 | 10 | 3.6 | 265 | 96.4 |

*Day *D. melanogaster* were introduced.
†Adults of this species were observed in the cage.

## DISCUSSION

It may be concluded that the coexistence of *D. melanogaster* and *D. funebris* in laboratory populations does not constitute an exception to the mathematical theories of Lotka and Volterra. If the populations are run in a closed system to which no food is added, one species, *D. funebris,* does eliminate the other. However, if the populations are run in an open system to which food is added at intervals, both species are able to survive. The relative proportions of the species will depend upon the condition of the food. In this case, the survival of both types is due to the fluctuations in the environment which favor first one species and then the other. This case differs to some extent from the work with graminivorous insects (Crombie, 1947) and Protozoa (Gause, 1934) where two species coexisted

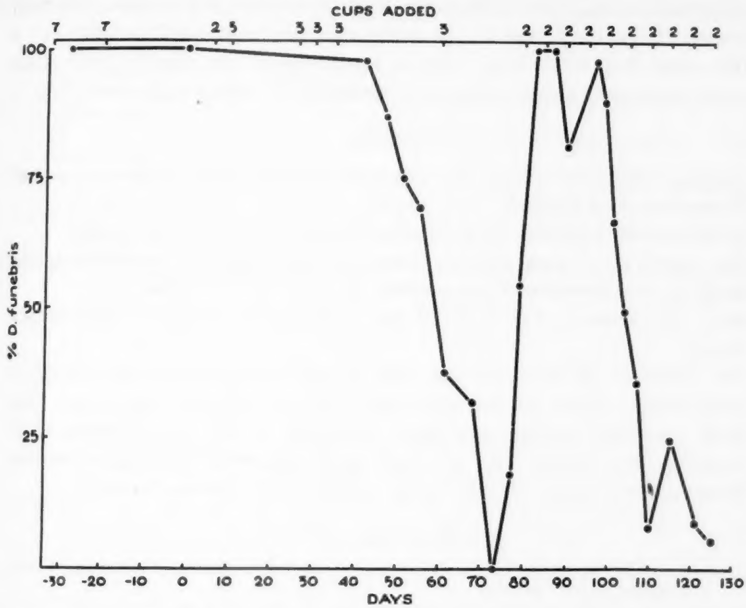


FIGURE 3. Competition between *D. melanogaster* and *D. funebris* in a population cage.

because they occupied different ecological niches. It is more comparable to the study of the polymorphic ladybird beetle, *Adalia bipunctata,* by Timofeef-Ressovsky (1940). The dark phase is favored during the summer months, but a greater proportion of the light phase survives hibernation. Thus, in that instance, the changing of the seasons favors alternate types and both survive.

Both *D. funebris* and *D. melanogaster* are cosmopolitan species whose wide distribution is evidence for their success under many conditions. *D. melanogaster* is generally regarded as a tropical species which has been introduced into many parts of its present range, but is absent from more northern regions and probably does not over-winter out-of-doors in some areas where it has been collected (Sturtevant, 1921). *D. funebris,* on the other hand, is probably primarily a temperate zone species (Timofeeff-Ressovsky, 1933) with a range extending farther to the north than that of *D. melanogaster.* Timofeeff-Ressovsky (1935) has shown that some of the success of *D. funebris* in various regions of the western Palaearctic can be attributed to the genetic adaptation of local populations to the temperatures prevailing in those regions. The work of Moore (1949a, 1949b) with the widely distributed leopard frog, *Rana pipiens,* has shown that similar adaptations make its distribution possible. It is conceivable that the success of many widespread species may be due not to some indeterminate innate superiority but to the ease with which their genotypes can be modified

by selection to adapt to a wide range of environmental conditions. However, although *D. funebris* and *D. melanogaster* are cosmopolitan species, it seems clear that in a given constant environment, one species will eliminate the other even though without competition the latter might survive.

## SUMMARY


1. Interspecific competition between populations of *D. melanogaster* and *D. funebris* was studied.
2. Both species coexisted in population bottles for almost two years.
3. The survival of both species does not constitute an exception to the mathematical theories of competition of Lotka and Volterra.
4. Analysis showed that most of the competition occurred between the larvae.
5. The survival of both species can be attributed to fluctuations in the environment which favored first one species and then the other. The fresh yeasted medium was more favorable to the development of *D. melanogaster* larvae. As the food aged and other microorganisms appeared, it was more suitable for the growth of *D. funebris* larvae.


## LITERATURE CITED


Bodenheimer, F. S., 1938, Problems of animal ecology. Oxford University Press, London. vii + 183 pp.

Crombie, A. C., 1947, Interspecific competition. J. Animal Ecol. 16: 44–73.

Darwin, C., 1859, The origin of species. Random House, New York. xvi + 1,000 pp.

Demerec, M., and B. P. Kaufmann, 1945, Drosophila guide. Carnegie Inst. Wash. 44 pp.

Dobzhansky, Th., 1947, Adaptive change induced by natural selection in wild populations of Drosophila. Evol. 1: 1–16.

Gause, G. F., 1934, The struggle for existence. Williams and Wilkins, Baltimore. ix + 163 pp.

L'Heritier, Ph., and G. Teissier, 1935, Recherches sur la concurrence vitale. Etude de populations mixtes de *Drosophila melanogaster* et de *Drosophila funebris*. C. R. Soc. Biol. 118: 1396–1398.

1937, Elimination des formes mutantes dans les populations de Drosophile. C. R. Soc. Biol. 124: 880–884.

Lotka, A. J., 1925, Elements of physical biology. Williams and Wilkins, Baltimore. xxx + 460 pp.

Lutz, F. E., 1911, Experiments with *Drosophila ampelophila* concerning evolution. Carnegie Inst. Wash. Pub. No. 143.

Moore, J. A., 1949a, Geographic variation of adaptive characters in *Rana pipiens* Schreber. Evol. 3: 1–24.

1949b, Patterns of evolution in the genus Rana. Chap. in "Genetics, Paleontology, and Evolution." Ed. by Jepsen, Mayr, and Simpson. Princeton University Press, Princeton.

Pearl, R., 1925, The biology of population growth. Alfred A. Knopf, New York. xiv + 260 pp.

Reed, S. C., and E. W. Reed, 1948, Natural selection in laboratory populations of Drosophila. Evol. 2: 176–186.

1950, Natural selection in laboratory populations of Drosophila. II. Competition between a white-eye gene and its wild type allele. Evol. 4: 34–42.

Ross, R., 1908, Report on the prevention of malaria in Mauritius. Waterlow and Sons, London. xvi + 202 pp.

Sang, J. H., 1950, Population growth in Drosophila cultures. Biol. Rev. (Cambridge Phil. Soc.) 25: 188–219.

Sturtevant, A. H., 1921, The North American species of Drosophila. Carnegie Inst. Wash. Pub. No. 301.

Timofeeff-Ressovsky, N. W., 1933, Über die relative Vitalität von *Drosophila melanogaster* und *Drosophila funebris* unter verschiedener Zuchtbedingungen, in Zusammenhang mit Verbreitungsarealen dieser Arten. Arch. Naturgesch., N. F. 2: 285–290.

1935, Uber geographische Temperaturrassen bei *Drosophila funebris* F. Arch. Naturgesch., N. F. 4: 245–257.

1940, Zur Analyse des Polymorphismus bei *Adalia bipunctata* L. Biol. Zentralbl. 69: 130–137.

Volterra, V., 1926, Variazioni e fluttuazioni del numero d'individui in specie animali conviventi. Mem. R. Accad. Nap. dei Lincei. Ser. VI, vol. 2 (Trans. in appendix of Chapman, R. N., 1931, Animal ecology. McGraw Hill, New York. x + 464 pp.

Zimmering, S., 1948, Competition between *Drosophila pseudoobscura* and *Drosophila melanogaster* in population cages. Amer. Nat. 82: 326–330.

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# "GLIDING" IN AMPHIBIANS AND REPTILES, WITH A REMARK ON AN ARBOREAL ADAPTATION IN THE LIZARD, ANOLIS CAROLINENSIS CAROLINENSIS VOIGT.

JAMES A. OLIVER

University of Florida and The American Museum of Natural History, New York

Thomas Belt (1874), in his interesting account of the natural history of Nicaragua, was impressed by the leap of a green lizard of the genus Anolis from a height of more than fifty feet. The lizard was being pursued by a green snake; both prey and predator came out of the tree in the same fashion. As soon as they hit the ground, the pursuit was continued without hesitation. The lizard had alighted on its feet and immediately hurried away.

The ability of arboreal forms to leap from great heights without harm is a valuable adaptation to life high above the surface of the ground. This ability reaches its maximum development among non-flying animals that have developed some form of a patagium and that are capable of gliding. Several phyletic lines are represented in this category of gliding forms. The best known examples among terrestrial vertebrates are: the Flying Squirrels (Subfamily Petauristinae), the Flying Phalangers (Genera Acrobates and Petaurus), and the Flying Lemurs (Genus Cynocephalus), among mammals; and the Flying Lizards (Genus Draco), among reptiles. A second category, less well-known, but of considerable interest, contains forms that possess small structural and behavioral modifications that enable the animals to retard the speed of descent through the air. With these forms descent is more rapid and at a lesser angle from the vertical line than is descent by gliding. In this category belong forms whose vernacular names erroneously imply a greater ability of aerial locomotion than is actually possessed; for example, the Flying Frog (several species in the Genus Rhacophorus) and the Flying Snake (Genus Chrysopelea).

These categories represent two extremely different degrees of arboreal adaptation and therefore it seems desirable to designate the two modes of descent by appropriate terms. In the past, the words "flying" and "gliding" have been used most commonly to describe aerial descent by non-flying vertebrates. The two terms have distinctly different meanings. Briefly, "flying" is aerial locomotion involving propulsion by means of the flapping or beating of wings, whereas "gliding" is aerial locomotion without any "motor" propulsion (i.e., beating of wings) wherein the animal loses altitude in order to maintain forward motion. Among living species of amphib-

ians and reptiles there are no species that can properly be designated as *flying* forms.

Other terms, such as volplane, plane, and soar, have also been used to describe the aerial movements of amphibians and reptiles. Hesse, Allee and Schmidt (1937) speak of structural "parachutes" and of "parachuting" forms. They state, "The essential principle involved is the development of rigidly supported membranes which enlarge the under surface of the body and permit gliding flight." This includes the mammals and the lizards mentioned in the first category above but excludes the forms named under the second category. Mertens (1948) employed the term "parachute fliers" (Fallschirmflieger) for the non-flying animals that can descend through the air from great heights without injury. He included under this heading both categories mentioned above. The term, parachute, is an accurate designation for the mode of descent described in the second category above.

Despite its broad usage to describe smooth movements that may be employed in locomotion through the air, on land, in water, or on ice, the term "glide" appears preferable to similar terms that might be employed to describe the first type of aerial locomotion given above. Here the emphasis in the use of these two terms is on the mode of descent rather than on structural modifications *per se*. On a purely arbitrary basis, the two modes of descent can be differentiated on the basis of the angle that the descent path takes from the vertical line. Thus *parachuting* can be considered as the making of a retarded descent along a path that deviates from the vertical by not more than $45^\circ$, whereas *gliding* is the performing of a retarded descent along a path of more than $45^\circ$ from the vertical. It is obvious that these two modes of descent will merge from one to the other, as do nearly all modes of locomotion in vertebrates. However, the two terms, as defined here, provide a convenient means of designating the differences in the degree of development of this arboreal adaptation. There are also differences in the degree of morphological specialization associated with the two modes of descent. For example, the gliding lizards (Draco) have a well-developed patagium that is supported by the elongated ribs. No such elaborately expanded and supported surface area is found in the parachuting forms.

Among amphibians and reptiles, the only forms definitely known to glide are the lizards of the genus Draco, of the tropical forests of southeastern Asia and the East Indies. The Fringed Geckos of the Genus Ptychozoon have been reported to glide but this now appears doubtful (Smith, 1935). These geckos, on the basis of the available accounts in the literature, would be referred to as parachuting forms. A number of other geckos of the Old World tropics and subtropics have probably developed this ability to some degree, for example, the Leaftailed Geckos of Madagascar (Genus Uroplates) and of Australia (Genus Phyllurus).

The only other recent reptiles that have been reported to show such adaptation are the tree snakes of the genera Chrysopelea and Dendrelaphis. The former of these includes the so-called "Flying Snakes." The mode

of descent observed in these is parachuting. This is accomplished by the snake leaping from a high limb, stretching out lengthwise, flattening and broadening the body, while drawing in the belly so that it forms a concave surface. In this manner these snakes have been observed to descend slowly and at an angle to the vertical.

Among amphibians only frogs have been reported to take to the air from great heights. Alfred Russel Wallace (1869) is responsible for one of the earliest reports on the parachuting ability of some members of the Genus Rhacophorus of the Indo-Malayan region. Several arboreal species of this Genus (*nigropalmatus* and *reinwardtii*) possess enormous webs between the fingers and toes, making a large expanse of skin surface relative to body weight. The aerial ability of these frogs has been much discussed. Ayyanger (1915) reported these frogs to be excellent gliders, whereas Barbour (1926) maintained that the ability of the frogs is greatly exaggerated. There seems to be little doubt that they are parachuters, rather than gliders, as the terms have been redefined here.

In Brazil, Cott (1926) performed some experiments that demonstrated a well-developed parachuting ability in a local tree frog, *Hyla venulosa.* When dropped from a height of 140 feet the frog landed ninty feet away from the vertical line! Cott later performed some experiments to evaluate the assertion that any small frog could probably duplicate this parachuting descent because of the greater surface area relative to body weight. He used a common terrestrial frog of Europe (*Rana temporaria*) and a European tree frog (*Hyla arborea*). The frogs were selected to approximate the size and weight of the Brazilian frogs. Both European species fell vertically and heavily. Moreover, there was an important behavioral difference between the Brazilian and the European frogs in falling. When the Brazilian frogs were launched into the air, they always stretched the limbs out at the sides in a constant and characteristic attitude, with the belly downward, even when tossed into the air in such a fashion that they turned over and over. Both European frogs turned over and over during the fall, and did not assume any constant position. It was thus purely chance whether they alighted on their feet or on their backs.

Mertens (1948) points out that the ability to glide or parachute is far more common among animals of the tropical rainforest than it is in those of any other habitat. This is especially true for amphibians and reptiles, and is, perhaps, to be expected in view of the greater numbers of arboreal species that occur in the rainforest as compared to none or only a few arboreal forms elsewhere. Certainly the most pronounced arboreal specializations to be found in amphibians and reptiles are present in the species of the tropical and subtropical areas: for example, the presence of a prehensile tail, adhesive disks, and stereoscopic vision.

Except for Cott's Brazilian Hyla, the only amphibians and reptiles that definitely have been reported to glide or parachute are inhabitants of the Old World tropics. It is to be expected that additional species inhabiting both the Old and the New World will be added to the list of forms now known to parachute.

## A PARACHUTING LIZARD

One of the previously unreported parachuting forms is the Carolina Anole, *Anolis carolinensis carolinensis* Voigt. This lizard has well-developed, expanded adhesive pads on the fingers and toes. With the aid of Edwin H. McConkey, I performed a series of simple experiments, to test the presence or absence of any ability to parachute in this lizard. Four lizards of approximately the same weight and size were dropped from a height of thirty-seven feet, two inches. They alighted six, six, ten and fifteen feet distant from the vertical line. The day was sunny and mild, with virtually no wind. The air temperature was 29°C, only slightly below the eccritic temperature of this species, 31.4°C.

One of the most interesting observations made in the experiments was that, in falling, all the specimens assumed a constant and characteristic posture with the limbs outstretched and the belly downward. Once this posture was assumed, there was no noticeable movement except for a slight wiggling of the tail. All the lizards fell vertically for one-third to one-half of the distance and then suddenly veered away from the vertical before striking the ground. In one instance the lizard alighted on concrete but was unharmed. From these experiments, the Carolina Anole seems to show a marked ability to parachute.

To obtain some comparative observations on another partly arboreal lizard of this same region, three Southern Fence Lizards, *Sceloporus undulatus undulatus*, were dropped from the same height and on the same day. These lizards were selected to approximate the weight or the length of the Anoles, but the species is much stockier and heavier. Two of the Fence Lizards were of the same weight as the Anoles but shorter; the third individual was of equal length but much heavier. Regardless of size, all the Fence Lizards alighted no farther from the vertical line than two feet and appeared to fall harder than the Anoles. The most striking difference noted was the fact that the Fence Lizards wiggled violently all the way down and did not assume any constant and characteristic posture; however, they appeared to alight on their feet.

In contrast to the Anole, the Fence Lizard does not appear to have a well-developed ability to parachute. Both species inhabit trees and bushes, at least part of the time. The Anole appears to be more arboreal than the Fence Lizard and is more frequently found at greater heights from the ground. The difference in ability to parachute in these two forms is probably due in large degree to the morphological differences and overall size. The proportion of surface area to body mass is considerably greater in the Anole than it is in the Fence Lizard. One of the Anoles had a snout-vent length of 65 mm and weighed 6.4 grams; a Fence Lizard of the same length weighed 15 grams. Fence Lizards lack expanded adhesive pads on the fingers and toes.

The Fence Lizards made no effort to assume and maintain while falling a constant posture with the arms and legs outstretched. This behavior was noticeably different from that of the Anoles in which a characteristic posture

was assumed by all of the individuals observed. This posture, with arms and legs outstretched and the belly downward, exposes the maximum possible surface, including the area of the expanded adhesive pads. It is of interest to note that the posture assumed by the Anoles is very similar to that reported by Cott for the Brazilian Hyla and for the parachuting Rhacophorus (see Cott, 1926, figs. 1-3). This is approximately the same position that is assumed by the gliding lizards (Draco), and the gliding mammals when in the air. Since these experiments, I have observed the same phenomenon in wild lizards under natural conditions. On several instances when I have pursued wild Anoles to the top of small trees they have endeavored to escape by leaping to the ground. Each time the lizard assumed the characteristic posture when in the air.

The evolution of this behavioral pattern in arboreal species appears to be of the utmost importance and is a constant characteristic of those forms that are known definitely to parachute. In the evolutionary development of the ability to parachute it appears that the acquisition of the behavioral pattern has proceeded independently of, and probably subsequent to, the development of structural modifications acquired for that purpose. All parachuting forms mentioned here differ considerably in their morphological attributes yet exhibit the same basic behavior pattern when parachuting. The morphological differences between the Brazilian *Hyla venulosa* and the European *Hyla arborea* are not sufficient to account for the vastly different performance recorded in Cott's experiments. Here the difference in performance results from a difference in behavior under the same circumstances.

It would be of considerable value and interest to study the differences in the ability to parachute in different species of a large genus containing arboreal and semiarboreal species in order to analyze the evolutionary development of the adaptive behavior patterns and various morphological adaptations associated with the arboreal habit. Among amphibians and reptiles, frogs of the genus Hyla and lizards of the genus Anolis would provide excellent material for such an investigation. In each of these genera there are highly specialized arboreal species, semiarboreal species, and terrestrial species. These differences in habits appear to be associated with well-marked differences in morphology and behavior, at least in the genus Anolis (Oliver, 1948). However, the actual analysis of these in relation to one another and in relation to phylogeny should prove a fascinating study and would considerably increase our understanding of the evolution of adaptive attributes.

## SUMMARY

The ability of some amphibians and reptiles to descend through the air from great heights without harm is reviewed. Among living species, two extremes of development of this arboreal adaptation are recognized on the basis of two different modes of descent: gliding and parachuting. The ability to parachute is reported for the Carolina Anole, *Anolis carolinensis carolinensis* Voigt.

## LITERATURE CITED

Ayyanger, M. P., 1915, A south Indian flying frog, *Rhacophorus malabaricus* (Jerdon). Rec. Ind. Mus. Calcutta, XI, 140-142.

Barbour, Thomas, 1926, Reptiles and amphibians, their habits and adaptations. Cambridge, Mass., xx +125 pp., figs. 1-142.

Belt, Thomas, 1874, The naturalist in Nicaragua. London, xvi + 403 pp., figs. 1-26, 1 map.

Cott, Hugh B., 1926, Observations on the life-habits of some Batrachians and Reptiles from the Lower Amazon: and a note on some mammals from Marajo Island. Proc. Zool. Soc. London, pp. 1159-78, pls. I-VI, figs. 1-3, 1 map.

Hesse, Richard, W. C. Allee, and K. P. Schmidt, 1937, Ecological animal geography. New York, xiv + 597 pp., figs. 1-135.

Mertens, Robert, 1948, Die Tierwelt des Tropischen Regenwaldes. Frankfurt, 144 pp., figs. 1-100.

Oliver, James A., 1948, The Anoline lizards of Bimini, Bahamas. Amer. Mus. Novitates, no. 1383, pp. 1-36, figs. 1-3, 2 tables.

Smith, Malcolm A., 1935, The fauna of British India, including Ceylon and Burma. Reptilia and Amphibia, II.-Sauria. London, xiii + 440 pp., 1 pl., figs. 1-94, 2 maps.

Wallace, Alfred Russel, 1869, The Malay Archipelago: the land of the orang-utan, and the bird-of-paradise. London, 2 vols., xxiv + 478 + 524 pp., figs. 1-51, 9 maps.

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# EFFECT OF TEMPERATURE AND MOISTURE CONTENT OF THE NUTRIENT MEDIUM ON THE VIABILITY OF CHROMOSOMAL TYPES IN DROSOPHILA PSEUDOOBSCURA

B. SPASSKY

Columbia University, New York

Most natural populations of *Drosophila pseudoobscura* are known to be mixtures of chromosomal types which differ in the gene arrangements in the third chromosomes. Observations in nature as well as laboratory experiments have shown that these chromosomal types differ in their adaptive values in different environments. Thus, at the temperature of 25°C, the heterozygotes for the Standard and Chiricahua gene arrangements are superior to the Standard homozygotes, and the latter are superior to the Chiricahua homozygotes. And yet, at 16°C the adaptive values of these chromosomal types are alike within the limits of the experimental errors (Dobzhansky, 1947). The influence of the humidity of the air on the survival rates of pupae, and of temperature on the longevity of the adult flies have been studied by Heuts (1947, 1948). The experiments reported below suggest that the moisture content in the culture medium in which the larvae develop also acts differentially on the survival of the different chromosomal types.

The material used consisted of eight strains, each homozygous for Standard, Arrowhead, or Chiricahua gene arrangements. The progenitors of these strains were collected at Pinon Flats, Mount San Jacinto, California, in 1944. These are the same strains which were used in the experiments of Dobzhansky and Heuts. Standard, Arrowhead, or Chiricahua flies were outcrossed to a laboratory strain which carried in its third chromosome the dominant gene Lobed and the recessive orange. The gene arrangement in the third chromosome marked with the gene Lobed was Santa Cruz; very little detectable crossing over takes place in the heterozygotes carrying Santa Cruz and the other chromosomes. In the $F_1$ generations of these crosses, Lobed/Standard, Lobed/Chiricahua, and Lobed/Arrowhead flies were selected and inbred in different culture bottles. Since Lobed is lethal when homozygous, a segregation in the ratio of 66.67 per cent Lobed: 33.33 per cent wild-type (homozygous for Standard, Arrowhead, or Chiricahua chromosomes respectively) should occur. Since, however, the gene Lobed reduces the viability of its carriers, the ratios actually observed vary, depending upon the viability of the different homozygous chromosomal

types. Observations on these ratios permit, therefore, comparisons of the viability performance of the Standard, Arrowhead, and Chiricahua homozygotes.

Two kinds of nutrient media have been used in the experiments. One of them, denoted as "Regular," has been described by Spassky (1943). It consists of 500 gm. of cream of wheat and 533 cc. of molasses in 3600 cc. of water. The "Dry" medium contained 500 gm. of cream of wheat and 400 cc. of molasses in 1800 cc. of water. Samples of both media were analyzed for water content. The regular medium contained about 72 per cent, and the dry medium about 54 per cent water.

Fifteen Lobed females and a like number of males served as parents of each culture. In all cases the females and males came from different strains with the same gene arrangement. This precaution is very important. Indeed we are interested in the relative viabilities of the Standard, Arrowhead, and Chiricahua flies as classes, and not in the viability of homozygotes for individual chromosomes, which may contain recessive viability modifiers, including lethals. The flies were permitted to oviposit for about twenty-four hours at room temperature, whereupon the adults were transferred, without etherization, into new oviposition bottles. The bottles with the eggs deposited in them were placed in constant temperature rooms either at 25°C or at 16°C. When the $F_2$ generation hatched, counts of Lobed and wild-type flies were made at 3-day intervals, until the cultures were exhausted. The viability data are summarized in table I.

No significant differences in frequencies of wild-type flies have been found among the homozygotes for the different gene arrangements developing on the normal medium at 25°C. At the same temperature, but on the dry medium the Chiricahua homozygotes survive relatively better than do Standard or Arrowhead homozygotes (the difference between Chiricahua and Standard is $3.44 \pm 1.62$ per cent in favor of Chiricahua, and that between Chiricahua and Arrowhead $6.6 \pm 1.46$ per cent in favor of Chiricahua. At 16°C, the only significant difference seems to be that between Standard and Arrowhead on regular food ($3.63 \pm 1.72$ per cent in favor of Arrowhead).

TABLE I

PERCENTAGES OF WILD-TYPE FLIES IN CULTURES WITH DIFFERENT GENE ARRANGEMENTS

| Gene arrangement | Regular medium | | Dry medium | |
|---|---|---|---|---|
| | Flies counted | Per cent wild-type | Flies counted | Per cent wild-type |
| Standard 25 | 1031 | 39.18 ±1.54 | 1557 | 37.44 ±1.23 |
| Chiricahua 25 | 1313 | 36.10 ±1.32 | 2096 | 40.88 ±1.07 |
| Arrowhead 25 | 923 | 35.31 ±1.57 | 2258 | 34.72 ±1.00 |
| Standard 16 | 1454 | 35.49 ±1.25 | 1597 | 36.01 ±1.20 |
| Chiricahua 16 | 2745 | 37.38 ±0.94 | 2844 | 34.38 ±0.89 |
| Arrowhead 16 | 1654 | 39.12 ±1.20 | 2589 | 34.80 ±0.94 |

More interesting for our purpose are differences in the viabilities of the same chromosomal types in different environments. Chiricahua homozygotes are relatively more viable on the dry medium than on the regular medium at 25°C (the difference is $4.78 \pm 1.70$ per cent). At 16°C Chiricahua homozygotes are more viable on regular than on the dry medium, the difference being $3.00 \pm 1.28$. Arrowhead homozygotes are more viable on the regular than on the dry medium at 16°C (the difference is $4.32 \pm 1.52$ per cent), although apparently not at 25°C. The Chiricahua homozygotes are relatively more viable at 25°C than at 16°C when raised on the dry medium (the difference is $6.50 \pm 1.39$ per cent), although no such difference is apparent on the regular medium.

It should be kept in mind that the present study is concerned with comparisons of the relative viabilities of the different chromosomal types during only a part of the life cycle, extending from the egg stage to the hatching of the adult. The viability, the fecundity, longevity, and other physiological characteristics of the adult flies are beyond the scope of the present study. The bearing of the present findings on the situation observed in natural and in experimental populations of *Drosophila pseudoobscura* is therefore limited. However, the characteristics studied are components of the overall adaptive values of the chromosomal types compared. As far as our data go, Chiricahua homozygotes seem to have certain survival advantages in warm and dry environments. It may be interesting in this connection to recall that the Chiricahua gene arrangement reaches its highest frequencies in the populations of Northern Mexico (Dobzhansky, 1948). On the contrary, the Arrowhead homozygotes may be superior in colder and more humid environment. The geographic distribution shows the highest frequencies of Arrowhead in the Great Basin, northwards up to the interior of British Columbia.[1]

## SUMMARY

The relative viabilities of the homozygotes for the Standard, Arrowhead, and Chiricahua gene arrangements have been compared in cultures with regular and with dry nutrient medium. The cultures were kept at temperatures of 16°C and 25°C. Chiricahua homozygotes find optimal conditions on the dry food and at 25°C, while Arrowhead homozygotes are superior on the regular food and at 16°C.

## LITERATURE CITED

Dobzhansky, Th., 1947, Adaptive changes induced by natural selection in wild populations of *Drosophila*. Evol. I: 1–16.

1948, Chromosomal variation in populations of *Drosophila pseudoobscura* which inhabit Northern Mexico. Amer. Nat. 82: 97–106.

[1]I wish to express my gratitude to Dr. Th. Dobzhansky for constructive suggestions.

Heuts, M. J., 1947, Influence of humidity on the survival of different chromosomal types in *Drosophila pseudoobscura*. Proc. Nat. Acad. Sci. 33: 210-213.

1948, Adaptive properties of carriers of certain gene arrangements in *Drosophila pseudoobscura*. Heredity 11: 63-75.

Spassky, B., 1943, Cream of wheat-molasses fly medium. Drosophila Information Service 17: 67-68.

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# A LIST OF CHEMICAL SUBSTANCES STUDIED FOR EFFECTS ON DROSOPHILA, WITH A BIBLIOGRAPHY[1]

IRWIN H. HERSKOWITZ

Louisiana State University School of Medicine

It was not until recently that the induction of heritable changes by chemical substances was clearly demonstrated in experiments with Drosophila by Auerbach and her associates. However, earlier work on the genotypic and phenotypic effects of various chemicals on Drosophila contain a wealth of information which may be of use to the investigator. There are several studies on chemically induced mutation which are not widely known, particularly those done in Russia by Rapoport, Gershenson, and others. Therefore, it was felt that a compilation of titles and a list of chemicals studied thus far in Drosophila would be useful.

The present handlist contains 314 references, including titles through the year 1950. Except for 50 new references, this work is almost identical with the one included by Dr. M. Demerec in Drosophila Information Service, No. 24: 107–122, 1950. In general the bibliography does not list papers concerned solely with ionizing and non-ionizing radiations, temperature, or age. A number of papers dealing with the lethal effect of insecticides on Drosophila have also been omitted. However, a few titles are included that show the variety of methods by which the induction of mutations has been attempted. References to studies on the development of a chemically defined medium for Drosophila are included since it is becoming apparent that more meaningful results will be obtained when the food variable is controlled.

The review articles by Timoféeff-Ressovsky (233) and Auerbach (10,11) have not been cross-indexed with the list of chemical substances. An asterisk in the list of chemical substances means that no test for mutagenicity has yet been made. Numbers following each substance refer to entries in the bibliography. An asterisk in the bibliography indicates that the reference contains no work on chemical mutagens; a question mark means that the paper was not available for checking.

## CHEMICAL SUBSTANCES

Acetic acid—142, 144, 172, 260
Acetylaminofluorine—64, 67, 74, 75
Acenaphthene—264

[1]This work was initiated at the Biological Laboratory, Cold Spring Harbor, N. Y., and completed in the Department of Surgery, L. S. U. School of Medicine where it has been supported by a grant from the National Cancer Institute, U. S. Public Health Service.

## BIBLIOGRAPHY

D. = Drosophila; D.m. = *Drosophila melanogaster;*
D. I. S. = Drosophila Information Service

* 1 . Alpatov, W. W., 1929, The influence of thyroid gland feeding on the acceleration of the growth of larvae of D.m. Proc. Nat. Acad. Sci. 15: 578–580.
2. Auerbach, C., 1940, Tests of carcinogenic substances in relation to the production of mutations in D.m. Proc. Roy. Soc. Edin., 60B: 164–173.
3. 1941, Tests of carcinogenic substances in respect of their influence on mutation in D.m. (Abstr.) Proc. 7th Int. Genet. Cong., Edin., 1939, J. Genet. suppl.: 51–52.
4. 1943, Chemically induced mutations and rearrangements. D.I.S., No. 17: 48.
* 5. 1943, Treatment of dechorionated eggs. D.I.S., No. 17: 66.
6. 1946, Chemically induced mosaicism in D.m. Proc. Roy. Soc. Edin. 62B: 211–222.
7. 1947, The induction by mustard gas of chromosome instabilities in D.m. Proc. Roy. Soc. Edin. 62B: 307–320.
8. 1947, Abnormal segregation after chemical treatment of D. Genetics 32: 3–7.
9. 1949, Tests of chemical substances for mutagenic activity. D.I.S., No. 23: 81.
10. 1949, Chemical induction of mutations. Proc. 8th Int. Genet. Cong., Stockholm, 1948, Hereditas suppl.: 128–141.
11. 1949, Chemical mutagenesis. Biol. Rev. Cambridge Phil. Soc. 24: 355–391.
12. 1949, Nuclear effects on chemical substances. Proc. 6th Int. Cong. Exp. Cytol., Stockholm, Exp. Cell Res. suppl. 1: 93–96.
13. 1949, The mutagenic mode of action of formalin. Science 110: 419–420.
14. 1950, Possible differences between the effects of chemical and physical mutagens. Pubbl. Staz. Zool. Napoli, suppl. 22: 1–21.
15. 1950, SH poisoning and mutation. Experientia 6: 17.
16. Auerbach, C., and H. Moser, 1949, Analysis of the genetical action of formalin-treated food. D.I.S., No. 23: 82.

17. Auerbach, C., and J. M. Robson, 1942, Experiments on the action of mustard gas in D. Production of sterility and of mutation. Report to the Ministry of Supply, W 3979.
18. 1942, Experiments on the action of mustard gas in D. II. Genetical differences in susceptibility. III. Proof that H acts directly on the chromosomes. IV. The production of visible mutations. Report to the Ministry of Supply, W 11831.
19. 1944, Production of mutations by allyl isothiocyanate. Nature 154: 81.
20. 1946, Chemical production of mutations. Nature 157: 302.
21. 1947, The production of mutations by chemical substances. Proc. Roy. Soc. Edin. 62B: 271–283.
22. 1947, Tests of chemical substances for mutagenic action. Proc. Roy. Soc. Edin. 62B: 284–291.
23. Auerbach, C., J. M. Robson, and J. G. Carr, 1947, The chemical production of mutations. Science 105: 243–247.
24. Bertani, G., 1947, Action of mutagenic substances. D.I.S., No. 21: 82.
25. Bhattacharya, S., 1948, Test for mutagenicity of methylcholanthrene. Nature 162: 573.
26. 1949, Tests for a possible action of ethylene glycol on the chromosomes of D.m. Proc. Roy. Soc. Edin. 63B: 242–248.
26a. 1949, A study of the inability of methylcholanthrene to produce mutations in D.M. Proc. Zool. Soc. Bengal 2: 187–193.
* 26b. Birch, M. M., 1944, The effect of arginine on larval growth in D.m. Thesis: Wellesley College, 49 pp.
27. Bird, M. J., 1949, (Mutagenicity of three compounds in N mustard class.) D.I.S., No. 23: 83–85.
28. 1950, Production of mutations in D. using four aryl-2-halogenalkylamines. Nature 165: 491.
28a. 1950, Mutagenic chemicals. D.I.S., No. 24: 78–79.
* 29. Bodenstein, D., 1947, Chemical alteration of development in D. (Abstr.) Anat. Rec. 99: 590.
* 30. Bodenstein, D., and A. Abdel-Malek, 1949, The induction of aristapedia by nitrogen mustard in *D. virilis*. J. Exp. Zool. 111: 95–115.
31. Born, H. J., and K. G. Zimmer, 1940, The influence of incorporated salts of heavy metals on the frequency of X-ray induced mutations. D.I.S., No. 13: 69.
32. 1940, Zur Frage der Steigerung der mutationsauslösenden Wirkung der Röntgenstrahlen durch Einbringung schweratomiger Salze in den Organismus. II. Z.i.A.V. 78: 246–250.
33. Braungart, D. C., and G. E. Ott, 1942, A cytological study of the effect of colchicine on D.m. J. Hered. 33: 163–165.
34. 1942, Colchicine-induced polyploidy in the fruit fly. Science Counselor 8: 105.
* 34a. Brown, M. J., 1934, Alcohol tolerance of D. An instance of adaptation to an environment lethal to most organisms. J. Hered., 25: 244–246.
* 35. Browning, I., 1949, The relation between potassium oxalate concentration and crossing over. Univ. Texas Pub. No. 4920: 57–62.
36. Buchmann, W., and I. Hoth, 1937, Versuche an D.m. über den Einfluss von Ferrum oxydatum saccharatum liquid. auf die Mutationsauslösung durch Röntgenstrahlen. Biol. Zbl. 57: 355–363.
37. Buchmann, W., and G. Sydow, 1940, Weitere Versuche an D.m. über den Einfluss von Schwermetallsalzen auf die Mutationsauslösung durch Röntgenstrahlen. Versuche mit Uranylacetat. Biol. Zbl. 60: 137–142.
38. Buchmann, W., and K. G. Zimmer, 1940, Zur Frage der Steigerung der mutationsauslösenden Wirkung der Röntgenstrahlen durch Einbringung schweratomiger Salze in den Organismus. I. Z.i.A.V. 78: 148–154.

39. 1941, Zur Frage der Steigerung der mutationsauslösenden Wirkung der Röntgenstrahlen durch Einbringung schweratomiger Salze in den Organismus. III. Z.i.A.V. 79: 192–198; also Arb. Reichsgesundh. 74: 411-417.
40. Burdette, W. J., 1949, The lethal mutation rate in D.m. following the administration of 20-methylcholanthrene and methyl-*bis* (betachloroethyl) amine hydrochloride. (Abstr.) Cancer Res. 9: 594.
41. 1949, Lethal mutation rate following serial administration of methyl-*bis* (betachloroethyl) amine hydrochloride. (Abstr.) Rec. Genet. Soc. Amer. 18: 79; also Genetics 35: 99, 1950.
42. 1950, Comparison of mutation rate and tumor incidence in D. treated with chemicals. (Abstr.) Rec. Genet. Soc. Amer. 19: 92.
43. 1950, Tumors and mutation in D. Texas Reports on Biol. and Med. 8: 123–133.
44. 1950, Studies on D. tumors. (Abstr.) Cancer Res. 10: 209.
45. 1950, Lethal mutation rate in D. treated with 20-methylcholanthrene. Science 112: 303–306.
46. Buzzati-Traverso, A., 1947, Sull' effecto mutageno del fosfato di sodio. Ricerca Scientifica e Ricostruzione 17: 1383–1384.
47. 1950, Perspectives of research on mutagens. (A discussion with the participants in the Symposium.) Pubbl. Staz. Zool. Napoli, suppl. 22: 171–186.
48. Buzzati-Traverso, A., and N. Visconti di Modrone, 1947, Produzione di mosiaci con rosso neutro in D.m. Ricerca Scientifica e Ricostruzione 17: 1128–1132.
49. Câmara, A., and S. de Vasconcelos, 1945, Não-disjuncão provocada artificialmente com andrido carbonico. Brotéria Ciênc. Nat. 14: 188–195.
* 49a. Carpenter, J. M., 1950, A new semi-synthetic food medium for D. D.I.S., No. 24: 96–97.
* 50. Carr, J. G., 1947, Apparatus for mustard gas treatment. Science 105: 369.
51. 1948, Chemically induced mutations. (Abstr.) Heredity 2: 283.
52. 1948, Chemically induced mutation. British J. Cancer 2: 132–134.
53. Chevais, S., and P. Manigault, 1942, Mutations experimentales provoquées par l'action d'un champ magnétique sur l'oeuf de D.m. C. R. Acad. Sci. Paris 214: 919–920.
54. Chevais, S., and J. A. Thomas, 1943, Variation du taux de mutations provoquées par l'0 aminophénylsulphamide, chez la Drosophile, suivant le delai compris entre le traitement du male et le croisement. C. R. des séances de la Soc. de Biol. 137: 187–189.
* 55. Child, C. M., 1942, Differential dye reduction in ovaries of *D. hydei.* Physiol. Zool. 15: 13–29.
* 56. 1943, Indophenol reaction and reduction in living ovaries of *D. hydei.* Physiol. Zool. 16: 141–161.
57. Child, G. P., 1946, The effect of delaying time of development on mutation rate. (Abstr.) Genetics 31: 214.
* 57a. Child, G. P., and T. Albertowicz, 1936, The effect of Nipagin on the wing size of vestigial of D.m. (Abstr.) Rec. Genet. Soc. Amer. 5: and Genetics 22: 188, 1937.
* 58. Cornman, I., 1940, Effects of ether upon the development of D.m. (Abstr.) Biol. Bull. 79: 347.
* 59. 1940, The effects of ether upon the development of D.m. (Abstr.) Collecting Net 15: 175.
* 60. 1943, Suspension of pole cell formation in D. eggs by hydrostatic pressure. J. Cell and Comp. Physiol. 22: 197–198.
* 61. 1944, The effects of ether upon the development of D.m. Physiol. Zool. 17: 367–377.

* 61a. Crozier, W. J., G. Pincus, and P. A. Zahl, 1936, The resistance of D. to alcohol. J. Gen. Physiol. 19: 523–557.

* 61b. Dantchakoff, V., and A. Vachkovitchuté, 1936, Sur l'immunité de la Drosophile envers les hormones sexuelles hétérologues. C. R. Soc. Biol. Paris, 121: 755–757.

* 61c. Delcourt, A., and É. Guyénot, 1910, De la possibilité d'étudier certains Diptères en milieu défini. C. R. Acad. Sci. (Paris) 151: 255–257.

* 61d. 1911, Variation et milieu. Lignées de Drosophiles en milieu stérile et défini. C. R. IV Conf. int. Génét: 478–487 (English summary).

* 61e. 1911, Génétique et milieu. Nécessité de la détermination des conditions; sa possibilité chez les Drosophiles. Technique. Bull. sci. Fr. Belg. 45: 249–333.

? 62. Demerec, M., 1947, Genetic potencies of carcinogens. (Abstr.) Fourth Int. Cancer Res. Congr., p. 43.

63. 1947, Mutagenic potencies of some polycyclic hydrocarbons and of some azo compounds. (Abstr.) Rec. Genet. Soc. Amer. 16: 29; also Genetics 33: 102, 1948.

64. 1947, Chemical induction of mutations in D. by the aerosol method. D.I.S., No. 21: 84.

65. 1947, Mutations in D. induced by a carcinogen. Nature 159: 604.

66. 1947, Production of mutations in D. by treatment with some carcinogens. (Abstr.) Science 105: 634.

67. 1948, Genetic potencies of carcinogens. Acta de l'Union contre le Cancer 6: 247–251.

68. 1948, Induction of mutations in D. by dibenzanthracene. Genetics 33: 337–348.

69. 1948, Mutations induced by carcinogens. British J. Cancer 2: 114–117.

70. 1949, Chemical mutagens. Proc. 8th Int. Genet. Congr., Stockholm, 1948, Hereditas suppl.: 201–209.

71. Demerec, M., R. Latarjet, S. E. Luria, E. F. Oakberg, and E. M. Witkin, 1946, The gene. Carnegie Inst. Wash. Yr. Bk. 45: 143–157.

72. Demerec, M., and S. E. Luria, 1945, The gene. Carnegie Inst. Wash. Yr. Bk. 44: 115–121.

73. Demerec, M., B. Wallace, and E. M. Witkin, 1948, The gene. Carnegie Inst. Wash. Yr. Bk. 47: 169–176.

74. Demerec, M., B. Wallace, E. M. Witkin, and G. Bertani, 1949, The gene. Carnegie Inst. Wash. Yr. Bk. 48: 154–166.

75. Demerec, M., E. M. Witkin, H. B. Newcombe, and G. H. Beale, 1947, The gene. Carnegie Inst. Wash. Yr. Bk. 46: 127–135.

* 76. Di Stefano, H. S., 1943, Effects of silver nitrate on the pigmentation of D. Amer. Nat. 77: 94–96.

77. Dotterweich, H., 1939, Über Beeinflussung der Mutabilität von D.m. durch Chemikalien. Zool. Anz. Suppl. (Verh. deutsch. zool. Ges. 41:) 12: 244–253.

78. 1940, Die Veränderlichkeit der Mutationsrate von D.m. nach generationenlanger chemischer Beeinflussung. Z.i.A.V. 78: 261–272.

79. 1941, Mutabilität und Umwelt. Versuche über den Einfluss von Follikelhormon auf die Mutationsauslösung bei D.m. Biol. Generalis (Wien) 15: 109–123.

80. 1943, Die Auslösung von Mutationen durch Follikelhormon und zusätzlichen Wärmeschock bei D.m. Biol. Generalis (Wien) 17: 419–446.

81. Dotterweich, H., and L. Schmidtke, 1941, Auslösung spezifischer Mutationen bei D.m. nach chemischer Dauerbeeinflussung. I. Versuche mit Follikelhormon. II. Der Einfluss hoher Progynon-Konzentrationen. Z.i.A.V. 79: 220–239.

* 81a. Duncan, F. N., 1915, An attempt to produce mutations through hybridization. Amer. Nat. 49: 575–582.

* 81b. Elwyn, A., 1917, Effect of humidity on pupal duration and on pupal mortality of D. ampelophila Loew. Bull. Amer. Mus. Nat. Hist. 37: 347–353.

? 82. Fábián, Gy., 1947, (Effect of colchicine injected into female D.) Arch. Biol. Hung. 17: 157–162.

83. Fábián, Gy., and G. Matoltsy, 1946, Test of a cancerogenic substance in respect to the "non-disjunction" frequency of the X-chromosome in D. Nature 158: 911–912.

84. 1947, The effect of 3–4 benzpyrene in respect to non-disjunction frequency in D.m. Arch. Biol. Hung. 17: 171–179.

84a. Fraile, A., and J. H. Sang, 1950, Induction of mutation with $Na_3PO_4$ and with Harmine. D.I.S., No. 24: 81.

? 85. Friedrich-Freska, H., 1940, (Carcinogens in D. and mutation.) Biol. Zbl. 60: 498.

? 86. Gelei, G. v., and L. Csik, 1939, (The effect of colchicine on D.m.) Magyar Biol. Kutatóintézet Munkái 11: 50–60.

87. 1940, Die Wirkung des Colchicins auf D.m. Biol. Zbl. 60: 275–286.

* 88. Gersh, E. S., 1946, Chemically induced phenocopies in D.m. D.I.S., No. 20: 86.

89. Gershenson, S. M., 1934, The effect of ether on gene changes. D.I.S., No. 1: 54.

90. 1939, The production of directed mutations with the aid of nucleic acid. Visti Akad. Nauk. USRR, 1939, No. 9–10: 83–84.

91. 1939, Induction of directed mutations in D.m. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 25: 236–238.

92. 1940, Types of mutations induced in D. by the sodium salt of thymonucleic acid. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 26: 601–611.

93. Gershenson, S. M., R. A. Zilberman, O. L. Levochkina, A. M. Pashkovski, P. O. Sitko, and N. D. Tarnavsky, 1947, (Induction of mutation in D. by dichlorodiethylsulphide.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 58: 1495–1496.

94. Gershenson, S. M., R. A. Zilberman, O. L. Levochkina, P. O. Sitko, and N. D. Tarnavsky, 1948, (Inducing mutations in D. with thymonucleic acid.) J. Gen. Biol. (Mosc.) 9: 69–88.

* 96. Gloor, H., 1946, Phenocopies of tetraptera and bithorax mutants. D.I.S., No. 20: 86–87.

* 97. 1947, Phänokopie-Versuche mit Aether an D. Rev. Suisse Zool. 54: 637–712.

* 98. 1949, Silver-reducing organ in D. larvae. D.I.S., No. 23: 89.

99. Goldat, S. J., and V. N. Beliaieva, 1935, (Artificial induction of mutations in D.m. through hydrochloric acid.) Biol. Zh. (Mosc.) 4: 379–384.

*100. Goldsmith, E. D., and M. H. Harnly, 1950, Reversal of the action of a folic acid antagonist, 4-aminopteroylglutamic acid, in D.m. (Abstr.) Cancer Res. 10: 220.

*100a. Goldsmith, E. D., M. H. Harnly, and E. B. Tobias, 1950, Folic acid analogs in lower animals. I. The Insecta: D.m. Ann. N. Y. Acad. Sci. 52: 1342–1345.

*101. Goldsmith, E. D., E. B. Tobias, and M. H. Harnly, 1948, (Folic acid antagonists and the development of D.m.) (Abstr.) Anat. Rec. 101: 743.

*102. Greiff, D., 1944, The effect of zinc sulfate upon fecundity and productivity of D.m. (Abstr.) Rec. Genet. Soc. Amer. 13: 20; also Genetics 30: 7–8, 1945.

*102a. Guyénot, É., 1913, Études biologiques sur une mouche, D. ampelophila Löw. I. Possibilité de vie aseptique pour l'individu et la lignée. C. R. Soc. Biol. Paris, 74: 97–99.

*102b. 1917, Recherches sur la vie aseptique et le développement d'un organisme en fonction du milieu. Thesis, Paris. 330 pp.

103. Hadorn, E., 1946, Mutationsversuche mit Chemikalien an D. I. Wirkung von Colchicin auf transplantierte Larven-Ovarien nach Behandlung in vitro. Rev. Suisse Zool. 53: 486–494.

104. 1950, Weitere Ergebnisse der "in vitro-Behandlung" von Drosophila-Ovarien mit Phenol. Pubbl. Staz. Zool. Napoli, suppl. 22: 32–49.

105. Hadorn, E., and H. Niggli, 1946, Mutations in D. after chemical treatment of gonads in vitro. Nature 157: 162–163.

106. Hadorn, E., S. Rosin, and G. Bertani, 1949, Ergebnisse der Mutationsversuche mit chemischer Behandlung von Drosophila-Ovarien in vitro. Proc. 8th Int. Genet. Cong., Stockholm, 1948, Hereditas suppl.: 256–266.

107. Hanson, F. B., 1935, Further data on the influence of physiological differences on the induced mutation rate: anesthesia, starvation and sex. Amer. Nat. 69: 211–222.

108. Hanson, F. B., and F. Hays, 1931, The relation between the induced lethal mutation rate and the conditions obtaining during treatment. I. Starvation. II. The influence of an anaesthetic. (Abstr.) Anat. Rec. suppl. 51: 121.

109. 1933, The relation of the induced mutation rate to different physiological states in D.m. II. Irradiation during complete anesthesia. Amer. Nat. 67: 419–428.

*110. Harnly, M. H., 1947, Quinine and cinchonine effects on growth, differentiation and lethal temperature of D.m. Amer. Nat. 81: 330–339.

*111. Harnly, M. H., and E. D. Goldsmith, 1946, The effect of thiourea on the development of D.m. (Abstr.) Anat. Rec. 96: 566.

*111a. 1950, Effects of thiourea, phenylthiourea, and propyl-thiouracil on mortality, growth, and differentiation of D.m. (Abstr.) Anat. Rec. 108: 564–565.

*111b. 1950, Mortality and thiourea resistance, 44 generations of D.m. (Abstr.) Anat. Rec., 108: 624.

*112. Hersh, A. H., E. Karrer, and A. L. Loomis, 1930, An attempt to induce mutation in D.m. by means of supersonic vibrations. Amer. Nat. 64: 552–559.

113. Herskowitz, I. H., 1947, A new method of treating D. gametes with chemicals. Evolution 1: 111–112.

114. 1947, Reports of summer investigators. 58th Ann. Report Biol. Lab., Cold Spring Harbor, N. Y., p. 24.

115. 1949, Tests for chemical mutagens in D. using the vaginal douche technic. Proc. Soc. Exp. Biol. & Med. 70: 601–607.

116. 1949, Tests for chemical mutagens in D. using the larval feeding technique. (Abstr.) Rec. Genet. Soc. Amer. 18: 93–94; also Genetics 35: 113–114, 1950.

117. 1950, The differential induction of lethal mutations by formalin in the two sexes of D. Science 112: 302–303.

*118. Hinton, T., and J. Ellis, 1950, A nucleic acid requirement in D. correlated with a position effect. (Abstr.) Rec. Genet. Soc. Amer. 19: 104–105.

*119. Hinton, T., J. Ellis, and D. L. Theriault, 1949, Amino acid and nucleic acid relationships in D. (Abstr.) Anat. Rec. 105: 513.

120. Horlacher, W. R., 1930, An attempt to produce mutations by the use of electricity. Science 72: 96–97.

*120a. Hutner, S. H., H. M. Kaplan, and E. V. Enzmann, 1937, Chemicals attracting D. Amer. Nat. 71: 575–581.

*121. Ingraham, A., 1944, Experiments with partial vacuum upon D.m. (Abstr.) Proc. Louisiana Acad. Sci. 8: 99–100.

*122. Kalina, B. F., 1950, Development and viability of D.m. on a medium containing DDT. Science 111: 39–40.

123. Kanellis, A., 1943, (Sex-linked lethals induced by X-rays after feeding oestron.) Z.i.A.V. 81: 191–195.

124. Kaplan, W. D., 1948, Formaldehyde as a mutagen in D. Science 108: 43.

125. Kaufmann, B. P., and H. Gay, 1948, The effect of near infrared radiation on the production of chromosome rearrangements in D. by nitrogen mustard. (Abstr.) Anat. Rec. 101: 20.

126. Kaufmann, B. P., H. Gay, and H. Rothberg, Jr., 1949, The influence of near infrared radiation on the production by nitrogen mustard of chromosome rearrangements in D. J. Exp. Zool. 111: 415–436.

127. Kaufmann, B. P., M. R. McDonald, H. Gay, N. C. Okuda, J. M. Pennoyer, and S. Blowney, 1949, Organization of the chromosome. Carnegie Inst. Wash. Yr. Bk. 48: 176–185.

128. Kaufmann, B. P., M. R. McDonald, H. Gay, K. Wilson, and R. Wyman, 1947, Organization of the chromosome. Carnegie Inst. Wash. Yr. Bk. 46: 136–146.

129. Kaufmann, B. P., M. R. McDonald, H. Gay, K. Wilson, R. Wyman, and N. C. Okuda, 1948, Organization of the chromosome. Carnegie Inst. Wash. Yr. Bk. 47: 144–155.

130. Keil, R., 1943, Der Kaliumgehalt der D.m. (Wildform) und die spontane Mutabilität. Biochem. Zeitschr. 313: 317–336.

131. Kerkis, J. J., 1935, (The effect of high frequency alternating potentials upon the production of mutations in D.m.) Trud. Inst. Genet. (Mosc.) 10: 223–226. (Russian with English summary.)

*132. Khouvine, Y., S. Chevais, and J. Grégoire, 1943, Action morphogénétique de dérivés de l'imidazol sur le mutant Bar de D.m. C. R. Acad. Sci. Paris 217: 161–163.

133. Kirsanov, B. A., and R. P. Martynova, 1947, (The effect of a 20-methylcholanthrene injection on the mutability of D.m.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 55: 765–769. (In Russian.)

*135. Koller, P. C., 1932, Der Einfluss chemisch reinen Thyroxins auf die Entwicklung von D.m. Arch. Ent. Mech. Org. 125: 663–672.

136. Kondakova, A. A., 1935, (Einfluss des Jods auf das Auftreten letaler Mutationen im II. Chromosom von D.m.) Biol. Zh. (Mosc.) 4: 721–726.

*136a. Koref, S., and D. Brncić, 1950, Influence of diethyl stilbestrol on D.m. tumors. D.I.S. No. 24: 83.

137. Kosiupa, D. E., 1936, The effect of sublimate on the occurrence of lethal mutations in D.m. Bull. Biol. Méd. exp. U.R.S.S. 2: 87–89.

138. Kossikov, K. V., 1935, The lack of effect of etherization on the X-ray mutation rate in D. simulans. Genetics 20: 363–365.

*138a. Lafon, M., 1937, Les facteurs liposolubles nécessaires à la croissance de D.m. Meig. C. R. Soc. Biol. Paris 124: 798–800.

*138b. 1937, Les facteurs hydrosolubles nécessaires à la croissance de D.m. Meig. C. R. Soc. Biol. Paris 124: 800–803.

*138c. 1939, Recherches sur quelques aspects du besoin qualitativ d'azote. II. Essai sur le besoin qualitativ d'azote chez un insecte: D.m., Meig. Ann. Physiol. et Physicochim. Biol. 15: 215–260.

139. Law, L. W., 1938, The effects of chemicals on the lethal mutation rate in D.m. Proc. Nat. Acad. Sci. 24: 546–550.

139a. Levochkina, O. L., 1940, (Nucleic acid induced mutation in the second chromosome of D.m.) Dopovidi Akad. Nauk., U.S.R.R., No. 3, 17.

*139b. L'Héritier, P., 1936, Sur l'apparition de différences de pigmentation entre diverses souches de D.m., type sauvage, à la suite d'une intoxication larvaire. C. R. Acad. Sci. (Paris) 203: 1392–1393.

*139c. L'Héritier, P., and G. Teissier, 1936, Carbon dioxide as a fly anesthetic. D.I.S., No. 6: 75.

140. Lobashov, M. E., 1934, (On the action of asphyxiation on the mutation process in D.m.) Trud. Leningr. Obsh. Estestv. 63: 271. (Russian with German summary.)

?141. 1934, (Ueber die Wirkung der Asfiktion auf den Mutationsprozess bei D.m.) Trud. peterfog. biol. Inst. 12: 371–378. (Russian with German summary.)

142. 1935, On the nature of the action of chemical agents on the mutation process of D.m. Candidate's Dissertation, Leningrad Univ.; also under title "Über die Natur der Einwirkung der chemischen Agentien auf den Mutationsprozess bei D.m." in Genetica 19: 200–241, 1937, and Trud. Leningr. Obsh. Estestv. 66: 345–376, 1937. (Russian with German summary.)

?143. 1948, (Study of reversible substantive change in the reproductive cells of D. by the method of vital dyeing.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 59: 557–560.

144. Lobashov, M. E., and F. A. Smirnov, 1934, (Über die Wirkung der Essigsäure auf Non-disjunction and Transgenationen bei D.m.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 2: 307–311. (Russian with German summary.)

145. 1934, (On the nature of the action of chemical agents on mutational process in D.m. II. The effect of ammonia on the occurrence of lethal transgenations.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S., 3: 174–178. (Russian and English text.)

*145a. Loeb, J., 1915, The salts required for the development of insects. J. Biol. Chem. 23: 431–434.

*145b. 1915, The simplest constituents required for growth and the completion of the life cycle in an insect (D.). Science 41: 169–170.

*145c. Luce, W. M., 1938, The effect of a 95 per cent oxygen 5 per cent carbon dioxide mixture upon facet number in infrabar in D.m. (Abstr.) Rec. Genet. Soc. Amer. 7: 79; also Genetics 24: 79.

*146. 1942, Effects of formalin upon facet number in the Bar alleles of D.m. (Abstr.) Genetics 27: 154–155.

147. Lüers, H., 1940, Experiments on the dependence of mutability upon body size. D.I.S. No. 13: 72.

*147a. 1950, Raising D. on a medium containing D.D.T. D.I.S. No. 24: 86.

148. Lüning, K. G., 1949, Gynandromorph production after mustard gas treatment. D.I.S. No. 23: 92.

149. MacDonald, J. M., 1948, Production of phenocopies in D. by chemical substances. (Abstr.) Heredity 2: 408.

150. Magrzhikovskaja, K. V., 1936, The influence of $CuSO_4$ on the mutation process in D.m. Bull. Biol. Méd. exp. U.R.S.S. 2: 90–92.

151. 1938, (The effect of $CuSO_4$ on the mutation process in D.m.) Biol. Zh. (Mosc.) 7: 635–642. (Russian with English summary.)

151a. Makino, S., and T. Mizuno, 1950, Some experiments to induce morphological aberrations with dinitrophenols and menthol in D. virilis. D.I.S. No. 24: 87.

152. Mann, M. C., 1923, A demonstration of the stability of the genes of an inbred stock of D.m. under experimental conditions. J. Exp. Zool. 38: 213–244.

*152a. Margolis, O. S., 1938, The effects of reduced atmospheric pressure and oxygen on facet number in bar-eyed D. (Abstr.) Rec. Genet. Soc. Amer. 7: 103.

?153. Martynova, R. P., 1948, (New experimental data on the mutative action of carcinogenic substances.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 60: 1569–1572. (In Russian.)

?154. Martynova, R. P., and B. A. Kirsanov, 1947, (The influence of carcinogenic substances on the mutation process in D.m.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 55: 647–650. (In Russian.)

*155. Matoltsy, G., and Gy. Fábián, 1947, (Measurement of the photodynamic effect of cancerogenic substances by biological indicators—D.) Arch. Biol. Hung. 17: 165–170.

156. Medvedev, N. N., 1935, (Artificial mutations under the combined influence of X-rays and salts of heavy metals in D.m.) Trud. Inst. Genet. (Mosc.) 10: 211-222. (In Russian.)

157. 1933, (Production of mutations by the combined action of X-rays and salts of heavy metals in D.m.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S.: 230–236. (Russian and English text.)

*158. Medvedeva, N. B., 1938, Action of folliculin on reproduction of coelenterata (Hydra) and insects (D.). J. Méd. Ukrain. 8: 197–208.

159. Milani, R., 1946, Induction of mutations with $Na_3PO_4$. D.I.S. No.20: 87–88.

160. 1946, Produzione di mutazioni letali in D.m. per mezzo fosfato di sodio. Sci. Genetica 2: 285–297.

161. 1946, Aumento della frequenza di mutazioni letali del chromosoma X di D.m. dopo trattamento con fosfato trisodico. Boll. Soc. Ital. Biol. Sperim. 22: 1173.

162. Mottram, J. C., 1930, The effect of carbon dioxide on the occurrence of non-disjunction in D. J. Exp. Biol. 7: 370–372.

163. 1930, Non-disjunction produced by carbon dioxide. Nature 125: 275–276.

164. Morgan, T. H., 1914, The failure of ether to produce mutations in D. Amer. Nat. 48: 705–711.

165. Muller, H. J., 1928, The production of mutations by X-rays. Proc. Nat. Acad. Sci. 14: 714–726.

166. 1930, Radiation and genetics. Amer. Nat. 64: 220–251.

167. 1941, Induced mutations in D. Cold Spring Harbor Symp. Quant. Biol. 9: 151–167.

168. 1946, Physiological effects on "spontaneous" mutation rate in D. (Abstr.) Genetics 31: 225.

169. Naumenko, V. A., 1936, Lethal mutations of D.m. induced by potassium permanganate. Bull. Biol. Méd. exp. U.R.S.S. 1: 204–206.

170. Nemeth, L., 1931, Untersuchungen über die Wirkung der Brenztraubensäure auf die D.m. Biochem. Zeitschr. 231: 385–392.

171. Niggli, H., 1946, Erzeugung von Mutationen mit Phenollösung durch Behandlung von Drosophila-Ovarien in vitro. Arch. Julius Klaus-Stift. 21: 475–479.

172. 1948, Mutationsversuche mit Chemikalien an D.m. II. Wirkung von Phenol bei Behandlung von Larvenovarien in vitro, sowie nach Verfütterung und Einbehandlung. Genetica 24: 79–160.

172a. Noujdin, N. I., 1946, (Thymonucleic acid induced mutation.) Bull. Exp. Biol. Med. 22: 38.

173. Olenov, J. M., 1940, The mutation process in D.m. under conditions of an avitaminous-$B_2$ regime. (In Russian, with French and English summary.) Vestn. Rentgenol. 22: 234–246.

174. 1941, The mutation process of D.m. under avitaminous $B_2$ conditions. Amer. Nat. 75: 580–595.

175. Ono, H., 1937, Induction of mutations by electric current. D.I.S. No. 7: 94.

*175a. Pearl, R., 1926, A synthetic food medium for the cultivation of D. Preliminary note. J. Gen. Physiol. 9: 513–519.

*176. Pearl, R., A. Allen, and W. B. D. Penniman, 1926, Culture media for D. II. A new synthetic medium and its influence on fertility at different densities of populations. Amer. Nat. 60: 357–366.

*176a. Pearl, R., and S. L. Parker, 1922, Experimental studies on the duration of life. III. The effect of successive etherizations on the duration of life of D. Amer. Nat. 56: 273–280.

*176b. Pearl, R., F. B. White, and J. R. Miner, 1929, Age changes in alcohol tolerance in D.m. Proc. Nat. Acad. Sci. 15: 425–429.

177. Pickhan, A., N. W. Timoféeff-Ressovsky, and K. G. Zimmer, 1936, Versuche an D.m. über die Beeinflussung der mutationsauslösenden Wirkung von Röntgen-und Gammastrahlen durch Hochfrequenzfeld (Kurzwellen) und Äthernarkose. Strahlentherapie 56: 488–496.

178. Ponomarev, V. P., 1937, (The effect of lead nitrate on mutation in D.m.) Biol. Zh. (Mosc.) 6: 69–80. (Russian with English summary.)

179. 1938, (The effect of $Pb(NO_3)_2$ on the process of mutation in D.m. II. Autosome mutations.) Biol. Zh. (Mosc.) 7: 619–634. (Russian with English summary.)

?180. Popoff, M., and A. Dimitrova, 1937, Durch chemische Einwirkungen experimentell erzeugte Mutationen bei D.m. Biol. gen. 13: 595–631.

?181. 1937, Über chemisch erzeugte Mutationen bei D.m. Z.i.A.V. 73: 467–469.

?182. 1938, Durch chemische Einwirkung experimentell erzeugte Mutationen bei D.m. Jb. Univ. Sofia, Med. Fak. 17: 81–119. (Bulgarian with German summary.)

*183. Poulson, D. F., 1947, Iron-accumulating cells in the larval gut of D. species. D.I.S. No. 21: 88–89.

184. 1949, Chemical differentiation of the larval mid gut of D. (Abstr.) Rec. Genet. Soc. Amer. 18: 110–111; also Genetics 35: 130–131, 1950.

*185. 1950, Physiological genetic studies on copper metabolism in the genus D. (Abstr.) Rec. Genet. Soc. Amer. 19: 118.

*185a. 1950, Influence of medium uptake and accumulation by larvae. D.I.S. No. 24: 91–92.

*186. Rapoport, J. A., 1939, Specific morphoses in D. induced by chemical compounds. Bull. Biol. Méd. exp. U.R.S.S. 7: 415–417.

187. 1940, Influence of thymonucleic and nucleic acids and some of their components on mutations. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 27: 1033–1036.

*188. 1940, On substances which disturb the symmetry in the organism. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 27: 369–372.

*189. 1941, (Phenocopy production.) J. Gen. Biol. (Mosc.) 2: 431.

?190. 1943, (Oxidation and mechanism of mutagenous factors.) J. Gen. Biol. (Mosc.) 4: No. 2.

191. 1946, Carbonyl compounds and the chemical mechanism of mutations. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 54: 65–67.

192. 1946, Polyploidy in animals induced by colchicine treatment of gonad rudiments. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 53: 67–70.

193. 1947, Derivatives of carbamic acid and mutations. Bull. Biol. Méd. exp. U.R.S.S. 23: 198–201.

?194. 1947, (Acetylization of gene proteins and mutation.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 58: 119–122.

195. 1947, (Reaction of free amino groups in gene proteins.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 56: 537–540.

?196. 1947, (Optically active substances and the symmetry of an organism.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 58: 1167–1170.

197. 1947, (Inheritance changes taking place under the influence of diethyl sulfate and dimethyl sulfate.) Doklady Vsesoyuz. Akad. Sel'sko-Khoz. Nauk im. V. I. Lenina 12: No. 10, 12–15.

198. 1947, (Chemical reaction with the protein amino groups in gene structure.) J. Gen. Biol. (Mosc.) 8: 359–379.

*199. 1947, On the synthesis of gene products in equimolecular quantities. Amer. Nat. 81: 30–37.

?200. 1948, (The alkylation of the gene molecule.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 59: 1183–1186.

201. 1948, (Effects of ethylene oxide, glycidol, and glycols on gene mutations.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 60: 469–472.

202. 1948, (Mutations under the influence of unsaturated aldehydes.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 61: 713–715.

*203. Reznitschenko, M. S., 1926, (The influence of the thyroid gland on the development of D.m.) Trud. Lab. exp. Biol. Mosc. Zoopark. 2: 181–200. (Russian with English summary.)

*203a. 1927, (Zur Frage über die spezifische Wirkung der Schildrüse und Ca- und K- ionen auf die Entwicklung von D.m.) Trud. Lab. exp. Biol. Mosc. Zoopark. 3: 27–35.

*204. Rosin, S., 1949, Chemically induced disturbances of bristle pattern. D.I.S. No. 23: 97.

*204a. Rubinstein, D. L., W. Lwowa, and H. Burlakowa, 1935, Über den Bedark des tierischen Organismus an Natrium und Calcium. (Versuche an D.) Biochem. Z. 278: 418–427.

205. Sacharov, V. V., 1932, (Erregung des Mutationsprozesses bei D.m. durch Jodbehandlung.) Biol. Zh. (Mosc.) 1(¾): 1–8. (Russian with German summary.)

206. 1933, (Auslösung von Mutationen bei D.m. durch Jodeinwirkung. II. Auslösung von Letalfaktoren.) Biol. Zh. (Mosc.) 2: 414–418. (Russian with German summary.)

207. 1935, (Jod als chemischer Faktor, der auf den Mutationsprozess von D.m. wirkt. III. Auslösung autosomaler Letalfaktoren.) Biol. Zh. (Mosc.) 4: 107–112. (Russian with German summary.)

208. 1936, Jod als chemischer Faktor, der auf den Mutationsprozess von D.m. wirkt. Genetica 18: 193–216.

209. 1938, On the specificity of the action of the factors of mutation. Biol. Zh. (Mosc.) 7: 595–618.

?210. Sacharov, V. V., and V. A. Naumenko, 1936, On the specifity (sic) of action of the mutation factors. II. Cyto-genetic analysis of lethals induced by chemical factors. Bull. Biol. Méd. exp. U.R.S.S. 2: 85–86.

*211. Schader, L., 1940, The effect of vitamin $B_1$ (thiamin) on the body and time of pupation of larvae of D.m. (Abstr.) Anat. Rec. suppl. 78: 39.

*212. 1941, The effect of vitamin $B_1$ (thiamin) on the body length and duration of the larval period in D.m. Growth 5: 19–26.

213. Schmitt, F. O., and C. P. Oliver, 1932, Electrostatic radiation and genetic effects. (Abstr.) Rec. Genet. Soc. Amer. 1: also Amer. Nat. 67: 67–68, 1933.

*214. Schultz, J., 1945, Reports of investigators. 56th Ann. Report Biol. Lab., Cold Spring Harbor, N. Y., pp. 38–39.

*215. Schultz, J., P. St. Lawrence, and D. Newmeyer, 1946, A chemically defined medium for the growth of D.m. (Abstr.) Anat. Rec. 96: 540.

*216. Schulze, P., 1912, Entwicklung von *D. rubrostriata* Becker in Formol; ein Beitrag zur Kenntnis der Lebensweise der Drosophilalarven. Zool. Anz. 39: 199–202.

217. Schütze, R., 1943, Die Beeinflussung der Mutationsrate bei D.m. durch kombinierte Behandlung mit Arsen und Röntgenstrahlen. Z.i.A.V. 81: 484–499.

*217a. Sekla, B., 1937, (Hereditary differences in the resistance to etherization in D.m. A contribution to the genetics of physiological characters.) Čas. Lék čes., Libenský Jubil. Vol. 76: 915–919. (Czech with English summary.)

?218. Shapiro, N. I., and I. Zholondz, 1935, (Data bearing on the influence of complete anaesthesia of the irradiated object with respect to the frequency of mutations induced by X-rays.) Zool. Zh. (Mosc.) 14: 311-316. (Russian with English summary.)

219. Slizynska, H., and B. M. Slizynski, 1943, The distribution of sex-linked lethals. D.I.S. No. 17: 65.

220. 1947, Genetical and cytological studies of lethals induced by chemical treatment in D.m. Proc. Roy. Soc. Edin. 62B: 234-241.

221. Slizynski, B. M., 1947, Production of structural changes in somatic chromosomes of D.m. Nature 159: 66-67.

*222. Stanley, A. J., G. H. Mickey, R. M. Melampy, and E. Oertel, 1943, Biological effects of the lipid fraction of royal jelly. Proc. Louisiana Acad. Sci. 7: 75-79.

223. Stekler, B. L., 1948, The use of diethylstilbesterol in the production of eye mutations in D.m. (Abstr.) Biol. Bull. 95: 258.

224. 1949, The use of diethylstilbesterol in the production of eye mutations in D.m. Commonwealth Bureau of Plant Breeding and Genetics. Plant Breeding Extracts 19: 244.

*224a. Stern, C., 1937, Methylene blue—staining of peripheral nervous system in D.m. D.I.S. No. 8: 90.

*224b. Sturtevant, A. H., 1939, High mutation frequency induced by hybridization. Proc. Nat. Acad. Sci. 25: 308-310.

*225. Svetlov, P. G., 1945, (Susceptibility of intestinal epithelium in males and females of D.m. to the noxious effect of lactic acid.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 48: 355-357.

*226. Svetlov, P. G., and O. V. Tschekanovskaja, 1945, (Sex differences in susceptibility of imaginal discs in D.m. larvae to noxious agents.) C. R. (Dokl.) Acad. Sci., U.R.S.S., N.S. 46: 293-295.

227. Takabe, K., 1944, Influence of supersonic sound waves upon heredity. I. Occurrence of "vestigial" in D. virilis. Dobutsugaku Zasshi (Zool. Mag.) 56: 43-47. (In Japanese.)

?228. Tarnavsky, N. D., 1938, A contribution to the question on the role of nucleinic acid in chromosome conjugation. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 20: 725-726.

229. 1939, On the role of nucleic acid in the production of directed mutations. C. R. Ukrainian Acad. Sci., Biology 1: 47-49.

229a. 1939, (Nucleic acid induced mutation.) Dopovidi Akad. Nauk., U.S.R.R., 47.

230. Thomas, J. A., and S. Chevais, 1943, Production expérimentale de mutation par les trois aminophénylsulfamides isomères chez la mouche D. Action sur les cellules males. C. R. des séances de la Soc. de Biol. 137: 185-186.

*231. 1944, Action tératologique élective du rouge neutre sur la mouche Drosophile en vie aseptique. C. R. Acad. Sci. Paris 219: 527-528.

*232. Thornton, D., 1947, The effect of 2,4-dinitrophenol on the larval growth of D.m. Growth 11: 51-60.

233. Timoféeff-Ressovsky, N. W., 1934, The experimental production of mutations. Biol. Rev. Cambridge Phil. Soc. 9: 411-457.

*234. Villee, C. A., and H. A. Bissell, 1947, Nucleic acids as growth factors in D. Biol. Bull. 93: 213 (Abstr.); also J. Biol. Chem. 172: 59-66, 1948.

*235. Villee, C. A., and M. D. Lowens, 1948, Barbituric acid as an inhibitor of growth and pupation in D. (Abstr.) Rec. Genet. Soc. Amer. 17: 61-62; also Genetics 33: 632-633.

236. Vogt, M., 1948, Mutationsauslösung bei D. durch Äthylurethan. Experientia 4: 68-69.

237. 1950, Analyse durch Äthlyurethan bei D. induzierter Mutationen. Z.i.A.V. 83: 324–340.

238. 1950, Ergänzende Befunde zur mutagenen Wirkung der Urethane (Carbaminsäureester) bei D. Z.i.A.V. 83: 341–346.

239. 1950, Urethane-induced mutations in D. Pubbl. Staz. Zool. Napoli, suppl. 22: 114–124.

240. Wenstrup, E. J., 1936, The effects of change in atmospheric pressure on mutation rate in D. (Abstr.) Rec. Genet. Soc. Amer. 5; also Genetics 22: 211–212, 1937.

*240a. Wheeler, B. M., 1947, The iodine metabolism of *D. gibberosa* studied by means of radioiodine $I^{131}$. Proc. Nat. Acad. Sci. 33: 298–302.

*241. 1948, A region of high iodine concentration in third instar larvae of *D. gibberosa*. (Abstr.) Anat. Rec. 101: 33–34.

*241a. 1950, Halogen metabolism of *D. gibberosa*. I. Iodine metabolism studied by means of $I^{131}$. J. Exp. Zool. 115: 83–108.

242. Whittinghill, M., 1948, The effects of methyl-*bis*(betachloroethyl) amine upon recombination values in D.m. (Abstr.) Rec. Genet. Soc. Amer. 17: 63–64; also Genetics 33: 634.

*243. 1950, Two crossover-selector systems: new tools in genetics. Science 111: 377–378.

*244. Wilson, L. P., 1940, The influence of sulfhydryl and sulfoxide on length growth of first instar larvae in D.m. Growth 3: 409–417.

*245. 1941, The influence of *l*-proline on larval growth in D.m. Growth 5: 11–18.

*246. 1942, Tolerance of larvae of D. for nucleic acid components: Adenine. Growth 6: 1–6.

*247. 1943, Tolerance of larvae of D. for nucleic acid components: Guanine. Growth 7: 273–278.

*248. 1943, Tolerance of larvae of D. for nucleic acid components: Uracil. Growth 7: 279–286.

*249. 1943, Tolerance of larvae of D. for nucleic acid components: Cytosine. Growth 7: 287–290.

*250. 1944, Tolerance of larvae of D. for nucleic acid components: Thymine. Growth 8: 117–124.

*251. 1945, Tolerance of larvae of D. for amino acids: Tryptophane. Growth 9: 145–154.

*252. 1945, Tolerance of larvae of D. for amino acids: phenylalanine, tyrosine and alanine. Growth 9: 341–352.

*253. 1946, Tolerance of larvae of D. for amino acids: methionine, cystine and cysteine. Growth 10: 361–373.

*254. 1949, The use of N-phenyl nile blue chloride as a vital stain for fat in D. D.I.S. No. 23: 103.

*255. Wilson, L. P., and M. M. Birch, 1944, Tolerance of larvae of D. for amino acids: Arginine. Growth 8: 125–148.

256. Witkin, E. M., 1947, Mutations in *Escherichia coli* induced by chemical agents. Cold Spring Harbor Symp. Quant. Biol. 12: 256–269.

*256a. Wolsky, A., 1937, (Die Wirkung des Kohlenoxyds auf den Sauerstoffverbrauch von Drosophila-puppen.) Math. term. Ért. 55: 879–890. (Hungarian with German summary.)

*256b. 1938, The effect of carbon monoxide on the oxygen consumption of D.m. pupae. J. Exp. Biol. 15: 225–234.

257. Zamenhof, S., 1942, Reports of investigators. 53rd Ann. Report Biol. Lab., Cold Spring Harbor, N. Y., pp. 30–31.

258. 1943, Proteolytic enzymes and mutations. (Abstr.) Rec. Genet. Soc. Amer. 11: 96; also Genetics 28: 96.

258a. 1943, Studies on factors influencing mutability. (Abstr.) Rec. Genet. Soc. Amer. 12: 57–58.

259. 1944, Effect of factors influencing mutability. Nature 153: 169.

260. 1945, Studies on induction of mutations with chemicals. II. Experiments with unstable genes. J. Genet. 47: 69–75.

261. Zamenhof, S., and M. Demerec, 1942, Studies on induction of mutations by chemicals. I. Experiments with heavy water (deuterium oxide). Amer. Nat. 77: 380–384.

262. 1943, Heavy water and mutations. (Abstr.) Rec. Genet. Soc. Amer. 11: 96; also Genetics 28: 96, 1943.

263. Zamiatina, N. D., and O. T. Popova, 1934, (Der Einfluss von Jod auf die Entstehung von Mutationen bei D.m.) Biol. Zh. (Mosc.) 3: 679–693. (Russian with German summary.)

264. Zivin, M. O., 1946, Effect of acenaphthene upon nondisjunction of sex chromosomes in D.m. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 52: 351–352.

?264a. 1946, (Influence of thymonucleic acid upon D.m.) C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 52: 263–264.

265. Zuitin, I. A., 1941, Influence of humidity contrasts on mutation rate in D. C. R. (Dokl.) Acad. Sci. U.R.S.S., N.S. 31: 927–929.

# DELETERIOUS GENES IN WILD DROSOPHILA MELANOGASTER FROM ISRAEL[1]

ELISABETH GOLDSCHMIDT

The Hebrew University, Jerusalem, and Columbia University

Since the concentration of unfavorable recessive genes in populations may furnish a clue to their hidden variability on the whole, and to their structure, size and breeding habits, it is important to gather a maximum of information on deleterious genes in populations of different geographic origin in a variety of species. Comprehensive investigations of lethal gene content in *D. pseudoobscura* were carried out by Dobzhansky and co-workers (1938, 1941, 1942). Recently, Brazilian populations of *D. willistoni* have been subjected to a similar analysis by Pavan et al. (1951). For a study of the variation in concentration of lethals under different environmental conditions, the cosmopolitan *D. melanogaster* should be a suitable object, but for this species only two sets of data from widely distant regions are available at present. While Ives's (1945) results from the United States indicate a very high concentration of lethal and semilethal genes (34–65 percent), Dubinin's (1945) figures for Russian populations are consistently lower, ranging from 9.8–29.2 percent, with the single exception of the Kutaisi population, which in November 1939, contained 38.9 percent lethals. Unfortunately, the results of these two authors are not strictly comparable, for it is not known to what level of viability semilethals are included in Dubinin's data.

However this may be, the population structure in the two areas investigated must be very different and it seems desirable to gain some information on the hidden variability of this species in other climatic regions. As a first contribution to an analysis of Middle East *D. melanogaster* populations, 243 second chromosomes from Israel were investigated by backcrossing to the *Cy L/Pm* stock, a modified 'Cl B method', which was described in detail by Wallace (1950a). Except for the few cultures, where not enough flies were available, five males and five females heterozygous for the same chromosome II and for the Cy L chromosome were utilized in the crucial $F_2$ cross. These five pairs were transferred twice, at intervals of three days, and the three bottles for each chromosome yielded an average of 552 flies, each bottle being scored at intervals of three days, three times, as a rule, until the appearance of the next generation. Figure 1 shows the frequencies of chromosomes of different viabilities, the latter being ex-

[1] This work was carried out during the tenure of the Mary E. Woolley International fellowship of the American Association of University Women, awarded by the International Federation of University Women.

pressed as the percentage of homozygous wild flies appearing in the total count for each chromosome. Disregarding crossing-over, the expected viability of the average heterozygote for two viable wild chromosomes II should be 33.33 percent, provided that the viability of the *Cy L* chromosome were normal. In reality, the viability of the *Cy L* chromosome utilized is reduced by somewhat less than one percent. The crossovers, on the other hand, which are more frequent among the offspring of young females and fall off to zero as the mothers grow older (cf. Whittinghill and Hinton, 1950), in their turn amount to a little below one percent. It is therefore most convenient to pool the crossover types with the curly-lobe class, and to determine the percentage of wild type homozygotes in the total, accepting 33.33 as the standard viability of the 'normal' heterozygote.[2] Accordingly, chromosomes of a viability less than 16.67 percent in the homozygous condition should be considered as semilethal. These figures compare well with those of Ives, who assumed the theoretical 33.3 percent for the average heterozygote and fixed 17 percent as the upper limit of the semilethal range.

The distribution of frequencies in figure 1 follows the characteristic pattern in showing two maxima, one at the lethal level (0–3.33 percent), and another one below the accepted normal level of 33.33 percent. This clustering of mutations at the lethal and at the subvital level and their comparative rarity throughout the intermediate range has been observed in all populations analyzed, wild and experimental, but developmental genetics has not furnished, as yet, a satisfactory explanation for this peculiar phenomenon.

The average viability of all chromosomes above the semilethal range amounts to 27.98 percent in the present sample. It is by no means surprising to find the average homozygote so much (5.35 percent) inferior to the average heterozygote and all workers concerned with recessive deleterious genes have considered this characteristic shift of the maximum as an indication of the importance of heterosis in the maintenance of populations.

The present analysis yielded 25.10 percent lethals (0–1.67 percent viability, corresponding to Pavan and co-workers' 5 percent viability level) and 13.58 percent semilethals, resulting in a total of 38.68 percent deleterious chromosomes. The lethals thus represent 64.89 percent of the total deleterious class. This proportion is in good agreement with Pavan and coworkers' data for *D. willistoni*, being intermediate between their percentage for the third (61.32 percent) and for the second (69.42 percent) chromosome, but it differs strikingly from the results of Ives (1945), who obtained 86.9 percent true lethals among his invisible deleterious genes, although he includes in the lethal class only those of complete inviability.

The sample from Israel analyzed here includes 41 chromosomes collected at different times in various parts of the country. The remaining 202 chromo-

[2] Dr. B. Wallace, Cold Spring Harbor, kindly informs me that this is the average viability of the heterozygote in several series of test crosses performed in his laboratory.

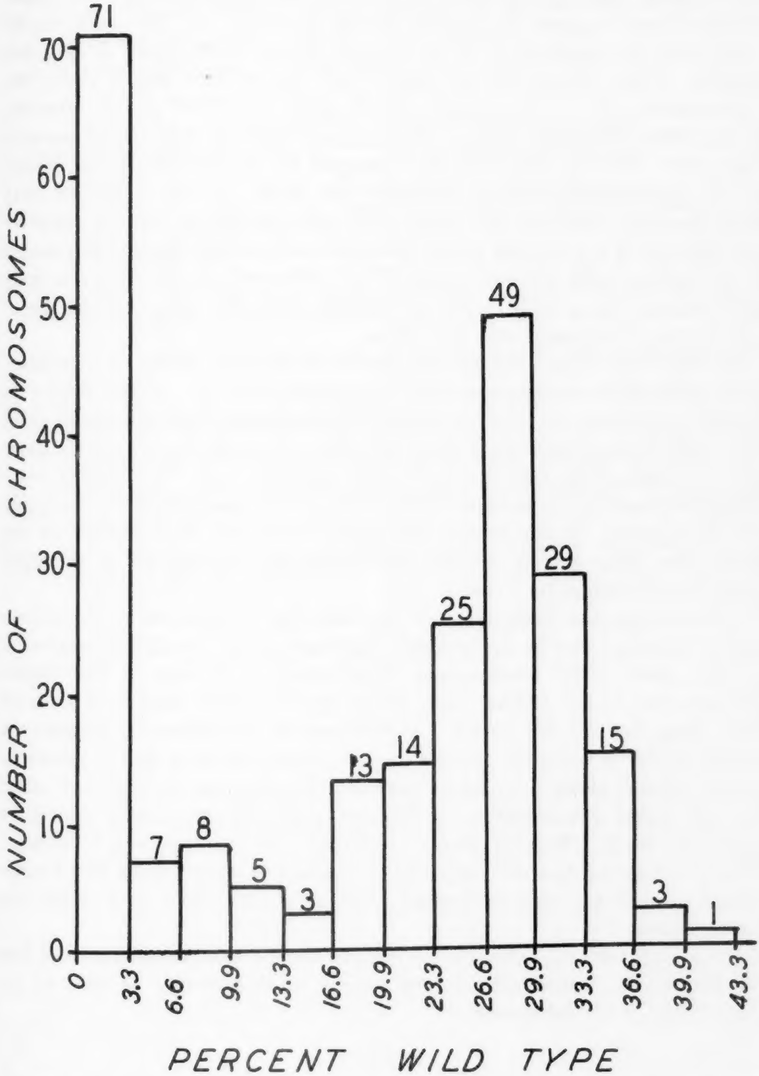


FIGURE 1. The frequencies of chromosomes of different viability classes in the total sample of 243.

somes came from two collections made by Mr. J. Wahrman in the mountainous Jerusalem area on October 20 and November 13, 1950, and may be considered to constitute a fairly uniform sample with regard to time and locality. The percentage of lethals and semilethals among these 202 chromosomes was 41.09 percent, a little above the 38.68 percent obtained for the total 243 chromosomes. This concentration of deleterious genes is comparable with the lowest figures obtained by Ives for the United States, being approximately midway between his value for Massachusetts, 1938 (45.0 percent), and that for Maine 1942 (34.2 percent). Ives's value for the viability of the average homozygote shows a certain inverse relationship to deleterious gene content; hence, it is interesting to note that the value here obtained is a little below his highest (28.53), indicating that population size at this range may be similar.

On the other hand, the percentage of deleterious genes in the Israel population of *D. melanogaster* is much higher than any of the values recorded by Dubinin for different Russian populations, with the exception of the Kutaisi population (38.8 percent). As stated above, we do not know what Dubinin defines as 'lethals,' but, assuming that he includes semilethals at least up to the 6.67 percent level, the comparable figures would be 32.1 percent for the total sample from Israel and 33.7 percent for the Jerusalem population in autumn and would still exceed all of Dubinin's percentages, except for Kutaisi.

This comparison indicates that the breeding populations of *D. melanogaster* in Israel may be fairly large, approaching the conditions reached in certain areas of the Northeastern United States. In view of the climatic peculiarities of the Middle East, where the cold rainy season is followed by a long, hot and dry summer, and in view of the difference between the colder and drier mountain climate and the hotter and more humid conditions of the coastal plain, it would be desirable to gain more specific information on the lethal concentration in different parts of the country during the seasonal cycle. While it stands to reason that the 'bottleneck' in population size in the Eastern United States should be at the close of the humid summer, it is not easy to predict when it should occur in a much more arid climate.

It is a pleasure to acknowledge the aid and encouragement received from Professor Th. Dobzhansky in the course of this study, as well as the hospitality of his laboratory.

## SUMMARY

An analysis of deleterious gene content in a sample of 243 second chromosomes of wild *D. melanogaster* from various parts of Israel yielded 38.68 percent lethal and semilethal genes; 202 chromosomes out of this total were collected in autumn in the Jerusalem area, and for this more uniform sample the content of lethals and semilethals was 41.09 percent. This concentration of deleterious recessives exceeds most of the values recorded by Dubinin for Russia, and is similar to his highest exceptional value; it

ranges among the lower percentages observed by Ives in populations of the Eastern United States. Hence in spite of the semi-arid climate, the size of the breeding populations of *D. melanogaster* in Israel may be comparable to that of certain areas in the humid Eastern United States.

## LITERATURE CITED


Dobzhansky, Th. and M. L. Queal, 1938, Genetics of natural populations. II. Genic variation in populations of *Drosophila pseudoobscura* inhabiting isolated mountain ranges. Genetics 23: 463–484.

Dobzhansky, Th. and S. Wright, 1941, Genetics of natural populations. V. Relations between mutation rate and accumulation of lethals in populations of *Drosophila pseudoobscura*. Genetics 26: 23–51.

Dobzhansky, Th., A. M. Holz, and B. Spassky, 1942, Genetics of natural populations. VIII. Concealed variability in the second and fourth chromosomes of *Drosophila pseudoobscura* and its bearing on the problem of heterosis. Genetics 27: 463–490.

Dubinin, N. P., 1946, On lethal mutations in natural populations. Genetics 31: 21–38.

Ives, P. T., 1945, The genetic structure of American populations of *Drosophila melanogaster*. Genetics 30: 167–196.

Pavan, C., A. R. Cordeiro, N. Dobzhansky, Th. Dobzhansky, C. Malogolowkin, B. Spassky, and M. Wedel, 1951, Concealed genic variability in Brazilian populations of *Drosophila willistoni*. Genetics 36. (In Press.)

Wallace, B., 1950, Autosomal lethals in experimental populations of *Drosophila melanogaster*. Evolution 4: 172–177.

Whittinghill, M. and C. W. Hinton, 1950, The nature of linkage variation with age in inversion heterozygotes of *D. melanogaster*. Proc. Nat. Acad. Sci. 36: 546–551.

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# NATURAL HISTORY AND THE BIOLOGICAL SCIENCES

CHARLES T. BRUES

Harvard University

The partially fire-proof and calamity-proof structure that houses workers in biology today has undergone a vast amount of remodeling. Since its first appearance as a tiny cyclone cellar which the blasts of dogma could not entirely erase, it has risen into the open quite in conformity with the heliotropic movement prevailing among those forms of animal and plant life that compete in the open. Its architecture has been kept continuously modern, for its time, and consequently much has been torn away, replaced—sometimes later to be resurrected—leaving here and there a few relics of its previous form. Of course, most of these have been polished, revarnished, or otherwise superficially disguised to suit the fleeting fancy of successive tenants.

Meanwhile, as in a pioneer New England dwelling, additional ells have been found necessary for the increasing family, including marital supplements—chemists, physicists, mathematicians, sociologists. The first generation crosses of these, all evincing the expected heterosis or hybrid vigor, include biochemists, endocrinologists, biophysicists, ecologists, geneticists, eugenicists, statisticians, to mention only a few conspicuous examples.

In later generations, naturally all these varied genes have popped up in many unexpected combinations. Certain polyploid agglomerations like the anthropologists confine their attention strictly to a single species of animal, though they allow nothing pertaining to his corporeal and spiritual body, nor his past history and unpredictable future, to escape their zealous eyes. Further afield, sociologists brazenly envision the rising tide of human depravity overwhelming man as a social animal, something for which he was not modeled by Nature. No less timid souls still further transcend the biological level. They seek by force of law to induce mutations in our instinctive and innate intellectual behavior to eliminate its unsocial traits. Perhaps entomologists may be to blame for this thesis, as they have surmised with good reason that significant mutations in instinct occur at long intervals among insects.

In contrast, other biological offspring, like the general physiologists, seek to compress into a few fundamental principles all the varied activities of the innumerable world of animate nature.

Thus, workers in one specialized field of biological science at the present day recognize the devotees of another with difficulty on the street, or in

the reports of their achievements before learned gatherings. These divergences began slowly, but the pace has been stepped up and the field of biology has been so unexpectedly widened and intensively cultivated in many circumscribed areas that the young naturalist who entered professional life half a century ago has seen the whole face of biological science change so completely that he can no longer see the forest for the trees. Furthermore, he is sufficiently familiar with so few of the trees thathe must be content to understand the remainder but vaguely on the authority of some colleague to whom the other denizens of the forest are equally intriguing, though unfortunately utterly strange.

This narrowness of understanding is the inevitable consequence of the biological structure itself. Those who have watched its modernization over an extended period of time have come to a gracious acceptance of their incompetence as mediators. They remain as outmoded experts in some rapidly narrowing corridor, frantically trying to keep track of their co-workers through the medium of some expansive rogues' gallery, "Who's Who," or by way of some short-cut to omniscience such as the *Biological Abstracts,* the *Zoological Record, or* other less encyclopedic digests.

Among the first groups of biologists to develop clannish propensities were the entomologists. They established numerous societies and journals that flourished during the last century, more luxuriantly after 1875. While this was going on, other zoologists continued collectively to pour their rapidly increasing output into a few more general journals, *Zeitschrifte* and *Jahrbücher.* When these finally blew up from internal pressure, the fragments congealed into an assortment of *Abteilungen* or separately named journals, variously devoted to more limited and discrete fields of research. To these entomologists are now contributing more and more extensively, together with others who find that insects are eminently suitable materials for varied kinds of experimental investigation.

With this background, it is refreshing to discern here and there on the far horizon some lone "naturalist" who still clings to the precarious supposition that the divergent phases of natural history are amenable to integration without loss of clarity or dignity. The exposition of such a viewpoint is contained in a recent book, entitled "The Nature of Natural History,"[1] by Marston Bates, an entomologist of wide and varied experience. This remarkably sane and well-balanced account of the varied aspects of natural history has furnished the occasion for the present remarks. Much that it details is commonplace to the professional biologist, but the vigorous exposition of natural history as a comprehensive background for the understanding of the several more restricted biological fields may be read universally and digested with profit. Such an outlook can do much to mitigate the insidious myopia that is creeping into teaching and research in the biological sciences.

[1] 309 pp. Charles Scribner's Sons, New York, 1950.